EFFECTS OF ENVIRONMENTAL AND FEEDBACK MANIPULATIONS ON EXERCISE REGULATION IN CYCLISTS

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i. ABSTRACT

The regulation of exercise, a process known as pacing, is considered an essential determinant of athletic performance. Pacing is the distribution of energy during an exercise task and manifests through pre-determined 'templates' built through prior experience and recalled for future performances. Once the exercise task begins, internal and external feedback is integrated to determine if the current work-rate should be maintained or adjusted. Despite a wide range of investigations examining the pacing phenomenon, the probability that environmental and feedback manipulations change pacing remains largely unknown. In addition, whether accurate or inaccurate knowledge of the fraction of inspired oxygen content (FiO_2) or series of oxygen deception trials can build psycho-physiological belief in cyclists to improve performance has not yet been explored. Moreover, whether an exercise reserve exists and can be used in hyperoxia to improve performance in hyperoxic time-trials (TT) and in subsequent normoxic TT's is unclear. Therefore, this thesis aimed to examine how environmental and performance feedback manipulations could affect exercise regulation and performance in trained cyclists.

A meta-analysis was conducted for Study One and the effects of five extrinsic factors on TT performance were examined: hypoxia, hyperoxia, heat-stress, pre-cooling strategies and various forms of feedback. Only studies that reported power output for self-paced cycling TT's in a manner that allowed for the segmentation of performances into start, middle and end sections were included. In Study Two, participants were informed during a series of 4000-m oxygen deception TT's they were inspiring either normoxia or hyperoxia, however, in two trials participants were deceived of the FiO_2. Trials were completed in a double-blinded and randomised order, allocating participants to one of four possible trial orders, before negative

performance deception was revealed in a final, self-paced normoxic TT. Participants in Study Three completed seven 4000-m TT's: two self-paced, normoxic familiarisation trials; a self-paced hyperoxic and competive hyperoxic trial; a further self-paced trial in normoxia, a hyperoxic competitive-performance deception trial; and a competitive, normoxic trial after deception was revealed.

The novel findings of this research were: i) self-paced TT's undertaken in hypoxia or heat-stress had no effect on cycling compared with normoxic or cool conditions until the middle section of trials where exercise intensity was downregulated (Study One); ii) provision of negative deceptive feedback may enable upregulation of MPO during the middle stages of a trial improving overall trial performance (Study One); iii) performance improvements in a previous negative deceptive feedback trial may be retained in a subsequent TT after negative performance deception is revealed (Study One); iv) cycling performance improves in hyperoxia, and mechanisms are likely physiological, however, improvement in a deception trial suggests a placebo effect might be present. (Study Two); v) a particular sequence of oxygen deception trials might build psycho-physiological belief to improve performance in a subsequent normoxic trial (Study Two); vi) further performance improvement can occur when receiving negative deceptive feedback in hyperoxia which indicates an exercise reserve still exists in hyperoxia (Study Three); and vii) performance improvements after negative performance deception in hyperoxia can be retained in a subsequent normoxic TT after deception is revealed (Study Three). These findings contribute to our knowledge of how exercise is modulated and offer mechanistic understanding of how trained cyclists can change their routine pacing strategy to enhance performance from exposure to increased FiO_2, oxygen deception trials and negative performance deception.

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vii. LIST OF ABBREVIATIONS

AIS	Australian Institute of Sport
CI	95% Confidence interval
CNS	Central nervous system
CON	Control
CO_2	Carbon dioxide
DSSQ	Dundee Stress State Questionnaire
FAMIL	Familiarisation time-trial
Fb	Feedback
FiO_2	Fraction of inspired oxygen
GE	Gross efficiency
GXT	Graded exercise test
HR	Heart rate
HNHN	Hyperoxic trials completed in order of: hyperoxic, normoxic, hyperoxic, normoxic
HNNH	Hyperoxic trials completed in order of: hyperoxic, normoxic, normoxic, hyperoxic
HYPER	Hyperoxic time-trial
HYPER-DEC	Hyperoxic deception time-trial (normoxic air inspired)
HYPER-PACER	Cyclists racing an on-screen pacer, representing the mean power output from a previous time-trial
HYPER-PACER-DEC102%	MPO of on-screen pacer increased by 2% during a hyperoxic time-trial
iEMG	Integrated electomyography

ICC	Intra-class correlation coefficient
Inform-fb	Informed feedback trial
INTER	Intervention
M	Mean
MD	Mean difference
min	Minutes
MVC	Maximal voluntary contraction
MPO	Mean power output
n	Sample size
N_2	Nitrogen
Neg-fb	Negative deceptive feedback
Neutr-fb	Neutral feedback
NHHN	Hyperoxic trials completed in order of: normoxic, hyperoxic, hyperoxic, normoxic
NHNH	Hyperoxic trials completed in order of: normoxic, hyperoxic, normoxic, hyperoxic
No-fb	No feedback
NORM	Normoxic time-trial
NORM-DEC	Normoxic deception time-trial (hyperoxic air inspired)
NORM-INFORM	Self-paced normoxic time-trial after deception was revealed
NORM-PACER104%	Competitive normoxic time-trial after deception with known pacer increase
P_{AER}	Aerobic energy contribution
P_{AN}	Anaerobic energy contribution
PEDro	Physiotherapy Evidence Database assessment criteria of study quality

PI	Pacing index
PO	Power output
Pos-fb	Positive deceptive feedback
PPO	Peak power output
PRIMSA	Preferred Reporting Items for Systemic Reviews and Meta-Analyses
O_2	Oxygen
RH	Relative humidity
RPE	Rating of perceived exertion
RPM	Revolutions per minute
S	Seconds
S_pO_2	Arterial (pulse) oxygen saturation
SD	Standard deviation
SEM	Standard error of mean
sEMG	Surface electromyography
SEQ	Sports Emotion Questionnaire
TT	Time-trial
UCRISE	University of Canberra Research Institute for Sport and Exercise
$\dot{V}O_2$	Minute oxygen consumption
$\dot{V}O_{2peak}$	Peak oxygen uptake
VL	Vastus lateralis (quadricep muscle)
W	Watts
$W{\cdot}kg^{-1}$	Watts per kilogram

1.0 INTRODUCTION

Athletic performance is largely dependent on an athlete's ability to maximise exercise intensity (i.e. velocity or power output) while minimising fatigue (116). To accomplish this, athletes attempt to maintain high levels of physical, psychological, technical and decision-making skills throughout an exercise task (93). The regulation of exercise intensity is a phenomenon in the sports science literature termed 'pacing'. Pacing is thought to be regulated by complex relationships between the brain and other physiological systems to avoid premature fatigue or competitive failure and ensure task completion (52). Therefore, understanding the mechanisms underpinning exercise regulation (pacing) may enable more informed decisions about how and where an athlete should best invest their energy during exercise to enhance performance.

Over the past thirty years, several models have been proposed to explain pacing phenomena including: the teleoanticipatory theory (166, 190), the central governor model (130), the perception based model (185), the pacing awareness model (52), and the psychobiological model (111, 131). In addition, recent reviews have suggested decision making (52, 118, 120, 146, 162) and neurophysiological determinants (147) are also important contributors to exercise regulation. Despite differing opinions, models and theories, the general consensus is that by repeatedly exercising the pacing strategy, pre-determined 'templates' are built from prior experiences and recalled for future performances (117). Although an initial pace is set based on the pre-determined template, the regulation of exercise across an exercise bout is then modulated based on afferent sensory feedback from various biological systems as the athlete responds to ongoing internal and external stimuli (70, 129, 130). Moreover, many of these models acknowledge the importance of factors such as performance goals and expectations (120), memory of prior experiences (117), knowledge of task duration (152, 173)

or current physical capacities (70, 117, 119, 129, 166), motivation and mood (44), and tactical considerations (146), in pace selection and overall performance success.

Based on previous research, a number of studies have shown physiological, psychological and environmental factors affect pacing (188). Controlled laboratory studies and field-based observations have reported an influence on pacing from: hydration status (50), carbohydrate (5) and caffeine ingestion (201), wind velocity (15, 172, 180), environmental terrain (29, 172, 196), other competitors (36), environmental and performance deception (89, 91, 158, 169, 171, 203), pacing feedback (183, 184), music (104), and self-talk (68). A focus in the literature has been the impact of different additional environmental stressors (e.g. heat-stress, hypoxia) on pacing and exercise performance. Therefore, interventions have been implemented to overcome the limitations of such environments including: acclimation or acclimatisation (34, 140, 145), pre-cooling (13, 49, 71, 102, 122, 123, 148, 153) and hydration practices (149, 167, 168). However, based on existing studies it is still difficult to determine what effect these challenging environments, and interventions to mitigate their limiting properties, have on overall performance and specifically, how exercise is regulated during exercise tasks.

Recently, researchers have provided deceptive perceptual, environmental or performance information via non-invasive and practical methods (204). The purpose of such deception is to change athletes' expectations and beliefs, such that they develop uncertainty in the pacing template and potentially deviate from their routine (pre-determined template) or planned pacing strategy (204). Despite several recent reviews on the influence of deception during exercise tasks (90, 204), the probability that differences in pacing strategy or performance are practically meaningful remains largely unknown.

Studies have delivered deceptive information via inaccurate performance information that misrepresents the athletes true performance (40). Of the different forms of deception, negative deceptive feedback, whereby athletes are informed that their performance is worse than reality, appears to be the most effective to elicit performance improvements (40). In previous negative deception trials, athletes have raced against a virtual on-screen pacer whom they believed was programmed to mimic mean power output (MPO) from their previous best performance. In reality, the pacer was programmed with a MPO which was 2% greater (40, 91, 158, 169). Performance improvements were believed to be the result of athletes accessing an exercise reserve, considered the remaining metabolic energy and proposed to be from finite anaerobic energy supplies (173). Despite recent advancements, the upper limit of these internal reserves, how they manifest, the options available for the body when utilising them (i.e. the proportion attributed to anaerobic or aerobic energy contributions) and techniques which exploit these capacities during competitive events warrant further research.

Another approach to alter task expectations and attempt to deviate athletes away from their routine pacing strategy is via environmental deception. A challenge for researchers providing deceptive environmental information is whether the deception is subtle enough to be effective. For example, previous research has examined the effects of deceptive information pertaining to ambient or core body temperature (31). A limitation of such an approach is that a change in ambient temperature is more likely to be detected due to thermal sensations and comfort, potentially revealing the deception (153, 156). Therefore, deceiving athletes of the fraction of inspired oxygen (FiO_2) may be more successful, as previous studies blinding athletes to the FiO_2 indicate even slight manipulations are sufficient to influence cycling performance (78, 87). For instance, previous research under hypoxic conditions indicates a

delay occurs from signalling peripheral receptors (i.e. blood chemoreceptors) before an adjustment to the pacing strategy (~30–60 seconds) is made (78, 87). However, to our knowledge, no current research describes the effects of FiO_2 deception on athletic performance. Therefore, whether knowledge of inspiring hyperoxia can build psycho-physiological belief and encourage an upregulation of exercise intensity compared to a normoxic trial, requires further exploration. Furthermore, despite earlier reports revealing hyperoxia permits a greater exercise capacity (10), it remains to be clarified whether a performance reserve exists in hyperoxia.

Currently, there is a lack of research surrounding environmental and feedback deception on pacing and performance. Moreover, until now, researchers have focused primarily on the influence of particular factors (i.e. hypoxia, heat) on pacing, and further, haven't considered the ergogenic effects of other forms of deception and the mechanisms underpinning changes, if any, to pacing. Therefore, the aim of this thesis is to explore how environmental and feedback manipulation affect exercise regulation in cyclists. This aim will be addressed by achievement of the following objectives (additionally, a visual flow chart representing the thesis conception and research questions is provided in **Figure 1.1** (page 6):

Objective 1 (Study One): To estimate the probability that a difference in pacing from environmental and feedback manipulations is practically meaningful. To achieve this, a comprehensive meta-analysis in cycling was conducted for this study.

Objective 2 (Study Two): To determine if a series of trials manipulating FiO_2 and belief (through deception of the composition of inspired air) might alter the pacing profile and performance during 4000-m cycling time-trials.

Objective 3 (Study Three): To investigate if an enhanced performance reserve exists when exercising in hyperoxic compared to normoxic conditions.

Objective 4 (Study Three): To determine if negative performance feedback could be used to change task expectations and build psycho-physiological belief such that performance in normoxia was improved. A cohort of trained cyclists were recruited as participants for this study.

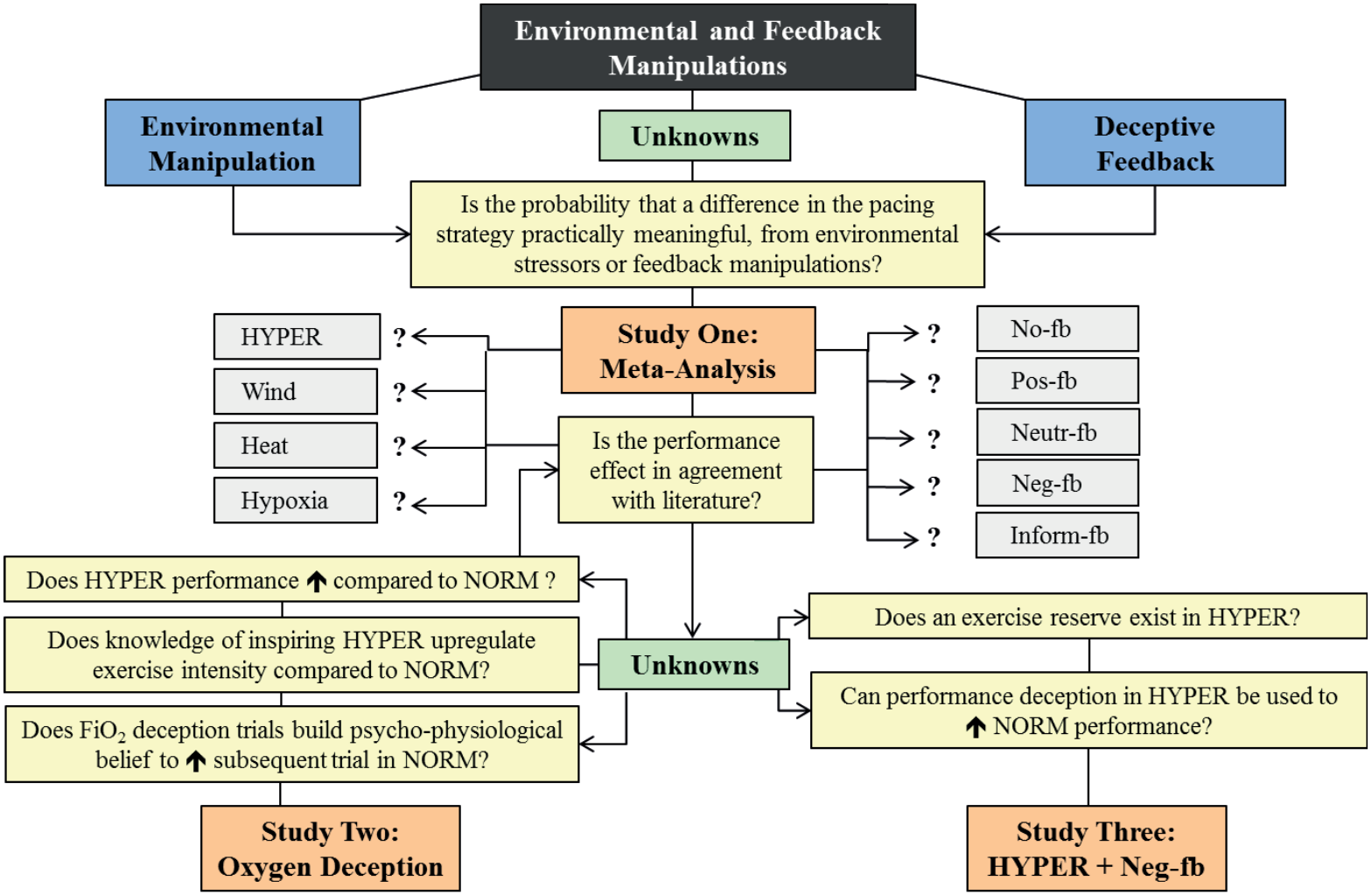

Figure 1.1 Visual flow chart on thesis conception and research questions. **?** represents an unknown in the current literature; ↑ indicates whether or not a performance improvement exists; FiO_2 = fraction of inspired oxygen; HYPER = hyperoxic trial; NORM = normoxic trial; Pos-fb = positive deceptive feedback; Neg-fb = negative deceptive performance feedback; Neutr-fb = neutral feedback; No-fb = no; Inform-fb = informed of the nature of a previous trial.

2.0 LITERATURE REVIEW

An athlete who can optimally regulate and distribute exercise intensity during competition is more likely to produce a best possible performance within individual psycho-physiological traits and environmental constraints on the day of performance. To maximise performance athletes must distribute energy or 'pace' according to their chosen event, while factoring intrinsic and extrinsic physiological, psychological and environmental variables (3, 164). In a cycling time-trial (TT) for example, these factors may influence an athlete's ability to execute their fastest time to completion while minimising the metabolic energy remaining at the finish line. (41, 192) It has been proposed that to achieve this, energy reserves – particularly the anaerobic energy reserve, should be fully expended by the end of a trial (41). However, it is vital the selection of pace is 'optimal' before reaching this critical point in the trial, where the energy reserve (or capacity) is not expended too far from the finish line, leading to premature fatigue and negatively impacting performance (182).

A multitude of sports, from individual events such as the 100-m sprint or marathon, to field-based team sports, require competitors to self-regulate their exercise intensity (3, 182). This phenomenon in the sport science literature is known as "pacing" and is characterised by "pacing strategies" or "pacing profiles" (61). Pacing is goal directed and regulated by complex relationships between the brain and other physiological systems about how and where an athlete chooses to invest their energy (52). Furthermore, athletic competition occurs in ever changing situations or environments. Therefore, what may be an optimal strategy during self-paced exercise, may not be appropriate during a competitive, 'racing' situation (3, 22). For example, in large mass start cycling events (such as The Tour de France), riders will implement or respond to various race tactics in the peloton or environmental conditions for the purpose of winning the race or days stage. Conversely, in a TT athletes 'race against the

clock' without direct interaction with competitors and the result is determined by the time to complete the given distance (61). Nevertheless, performance in both formats will be determined by an athlete's ability to appropriately respond to intrinsic and extrinsic physiological, psychological and environmental factors on the day of performance.

A major focus in the research literature has been to understand the mechanisms of pace regulation in response to a range of environmental conditions, these include: oxygen availability (10, 34, 142, 187), heat-stress (134), wind velocities (15, 172, 180), and environmental terrain (29, 172, 196). In particular, a large number of studies have investigated pacing and performances in hypoxia or during hot and humid conditions, as both are common environments athletes may be exposed to during training and competition (40, 159). In addition, the provision of various forms of feedback to athletes during exercise has provided important insight about how exercise is regulated based on a pre-determined template, knowledge and motivations or task expectations (204).

The aim of this review is to complement the comprehensive meta-analysis provided in Chapter 3, which describes the effect of environmental and feedback interventions on pacing profiles in cycling. As a result, specific areas not addressed in the meta-analysis but important to the context of this thesis will be explored in this chapter. Therefore, the following review addresses the nature of pacing in the context of self-paced exercise, providing an overview of the literature relating to the mechanisms and current perspectives on the physiology and psychology underpinning these mechanisms, and how these mechanisms manifest during performance, specifically, when environmental factors and incoming feedback are manipulated or withheld.

2.1 PACING AND PERFORMANCE

2.1.1 Pacing Strategies: Types and Characteristics

In a review by Abbiss and Laursen (3), six types of pacing strategies typically observed in sport were identified: all-out, positive, even, negative, parabolic and variable pacing. A visual representation of the various forms of pacing profiles is provided in **Figure 2.1 to 2.3,** and further detail of these identified strategies will be discussed in the following sections.

Figure 2.1 Types of pacing strategies adopted by athletes: positive pacing (**a**); negative pacing (**b**); even pacing (**c**); variable or parabolic pacing (**d**). From Abbiss and Laursen (3).

All-Out Pacing Strategies

The scientific literature demonstrates during very short duration events (< 30–60 seconds), athletes will typically adopt an all-out pacing strategy (25, 193). Athletes accomplish this by working at their maximal rate from the start of the event to accelerate and reach peak velocity as fast as possible, before attempting to maintain work output and kinetic energy to the finish line (**Figure 2.2**) (182). However, if the time spent accelerating to reach peak velocity is too

slow, all-out pacing can result in slower average speeds and a suboptimal performance. Consequently, all-out pacing may result in negative-splits, where despite high power outputs (PO) required to alter the kinetic energy from rest, the lower relative speeds in the initial start to peak velocity may consume a significant proportion of the event. As a result, the second half of the exercise bout may be completed in less time than the first (41, 61, 192). In addition, any metabolic energy left in reserve when crossing the finish line is essentially wasted since it can no longer be used to improve the performance (41, 192). Observations of an all-out strategy in athletes indicate performance times are dependent on whether the sport is water or air resisted and the type of locomotion involved (12, 15, 58, 172, 180, 183, 184). Specifically, in sports such as swimming and running, an all-out strategy lasting beyond 30 seconds would be less effective because a greater amount of energy is lost to frictional resistance (3), where the deterioration of velocity would become too great resulting in suboptimal performance times. This is particularly an issue in swimming where an increase in velocity results in greater drag resistance due to the higher drag properties of water compared to air (182). Comparatively, an all-out pacing strategy appears to be advantageous to cycling and speed skating lasting up to 60 seconds, where the resistive forces are lower (3, 41, 193). Therefore, though rapid fatigue will occur, the slowdown in velocity is not as pronounced (41, 192). An example is the 1000-m track cycling event which takes approximately 60 seconds to complete. Mechanical PO required to reach peak velocity is far greater compared to maintaining it, despite reductions in PO thereafter due to a rapid increase in fatiguing metabolites as the event progresses (**Figure 2.2**) (192). Collectively, current evidence suggests that successful performance in exercise lasting up to 60 seconds is achieved by reaching peak speed in the shortest possible time, and therefore, maximising time spent at peak velocity to record the fastest time to completion.

Figure 2.2 Example of power output and velocity profiles during a 1000-m track cycling event. Note the high power output needed to alter the kinetic energy from rest, and the gradual decline in velocity beyond 40% (~ 25 seconds) of the overall trial time. From Abbiss and Laursen (3).

Positive Pacing Strategies

A positive pacing strategy is common in both short and long form events, where the starting exercise intensity (i.e. PO) is near maximal and diminishes over the course of the exercise bout (**Figure 2.1a**) (3). Moreover, the nature of certain locomotive events, such as swimming (183), relay athletics (150) and rowing (65) events, involve specific starting procedures which allow peak velocity or that encourage velocities to be achieved in a shorter time. For example, dive starts in swimming or a flying start in athletic relay events, respectively (182).

Findings by Sandals et al. (150) suggested a further advantage of a positive pace selection in 800-m treadmill running is the ability to significantly increase oxygen uptake ($\dot{V}O_2$), a result in agreement with earlier findings in elite rowers during a 2000-m rowing TT (65). Therefore, a positive pacing strategy appears to minimise energy left in reserve when crossing the finish

line and make maximal use of athletes physiological capacities. However, the trade-off of a fast starting strategy, is an increased accumulation of fatigue related metabolites and higher ratings of perceived exertion (RPE) (183, 184). This is likely the result of a greater contribution from the anaerobic energy system associated with fast-twitch, or Type-II muscle fibres, which lack fatigue resistant qualities (182). Consequently, a reduction in exercise intensity is likely to occur, believed to be in response to incoming sensory feedback from the periphery, as a protective mechanism to prevent performance failure (8, 70).

In events lasting two hours or longer, a relatively fast starting strategy may be implemented as part of race tactics or based on individual goals, aims or expectations (3). In cycling for example, individual or small group breakaways from the main group (peloton) are common, as cyclists attempt to gain a competitive advantage (51). Moreover, Abbiss and Laursen (3) proposed athletes may inappropriately adopt a positive pacing strategy due to unrealistic expectations or ambitious perceptions regarding current abilities, and consequently, such tactics are often misguided and the chosen intensity too great compared to what can be tolerated. Therefore, a progressive reduction in exercise intensity may occur due to premature fatigue (3). However, positive pacing approaches are sometimes successful and as such, a positive pacing strategy has been suggested to be a viable tactic to employ in athletic competitions (183).

Negative Pacing Strategies

A negative pacing strategy is where an increase in exercise intensity occurs over the duration of an event resulting in negative-splits, where the second half of the event is completed faster than the first (**Figure 2.1b**). It is thought implementing such a pacing strategy can prolong exercise performance by sparing the rate of carbohydrate breakdown as an energy source, decrease the anaerobic energy use and limit accumulation of associated fatigue related

metabolites early in the exercise bout (3). However, despite this suggestion, experimental evidence of whether negative pacing is an effective strategy is limited. Fukuba and Whipp (63) suggested performance is compromised if an athlete starts too slowly, regardless of whether they attempt to make up for lost time, as metabolic limitations (i.e. accumulation of inorganic phosphate, potassium and hydrogen ions) may restrict their ability to do so. Furthermore, research by Thompson et al. (183) demonstrated a negative paced breaststroke swimming TT did not improve overall performance, despite a lowered heart rate (HR) response and similar $\dot{V}O_2$ values to even and positive paced TT's. Contrary to Thompson and colleagues (183) findings, Mattern et al. (114) noted improved performance in a 20-km cycling TT when trained cyclists were forced to start at a PO 15% lower compared to self-paced trials. The authors attributed this finding to significantly lower blood lactate concentrations during the first nine minutes of the negative-paced trial. Nevertheless, further research suggests adopting a negative pace may lead to an underperformance, particularly for exercise durations of 30 to 40 minutes where the exercise intensity is severe (63, 182). However, negative pacing is often observed in the final stages of individual self-paced TT's where athletes increase work-rate with closer proximity to task completion; a phenomenon in the pacing literature known as an 'end spurt', which has been shown to be influenced by knowledge of the remaining exercise distance or duration (3, 17, 166). The performance observation of an exercise end spurt is described further in Section 2.3.2.

Even Pacing Strategies

An even pacing strategy (**Figure 2.1c**) is characterised by a constant distribution of pace across a known exercise distance or duration (182). During prolonged events (> 2 minutes) when environmental conditions are stable, an even pace may be appropriate for events such as running, swimming, rowing, cross-country skiing, speed skating and cycling (3, 41, 183, 184). In particular, events where drag resistance properties can predominately be a limiting factor in

energy preservation, minimising variations in pace can be critical (3). Therefore, in events susceptible to greater drag resistance, mitigating fluctuations in acceleration and deceleration can be advantageous to reduce the energy cost of the event and optimise performance (3, 182). Moreover, minimising variations in pace for events longer than two hours in duration may help to reduce depletion of energy stores and the onset of early fatiguing milieu associated with psychological and physiological fatigue (54, 77, 97, 100). In contrast, during a relatively controlled setting, Wilberg and Pratt (200) observed that more successful cyclists in the 1000-m to 4000-m track events implemented an even pacing strategy compared to less successful riders. More recently, Thomas et al. (181) provided insight into mechanisms underpinning implementation of an even pacing strategy, where reduced blood lactate concentrations, perceived effort and non-significant trend for lower respiratory exchange ratio were found compared to self-paced and variable pacing strategies in 20-km cycling TT's. In contrast, Lander and colleagues (98) dispute against enforcing a constant pacing strategy as it poses a significantly greater physiological challenge in comparison to a self-selected pace. The author's explanation is even pacing limited rowers ability to choose or dictate the pace, where fluctuations in work-rate are believed to be an important behavioural response to regulate exercise (98). Evidence of this behaviour has been observed in the pace distribution in elite marathon runners, where negative pacing or variable pacing has been proposed the most efficient way for athletes to achieve their best performance and prevent premature fatigue (121). Overall, it appears several intrinsic and extrinsic factors influence whether or not an even pace selection is appropriate to achieve the best possible performance.

Parabolic Pacing Strategies

The recent advances in technology now permit high frequency (i.e. second by second) measurement of performance metrics such as velocity and PO, providing greater resolution for analysing pace selection. As a result, further insight into where and how the distribution of

energy occurs across an event can be examined, instead of simply reporting pace selection during the first or second half of an exercise bout. Consequently, athletes will often start with an initial high velocity during exercise tasks over longer durations, reducing exercise intensity in the middle portion, before increasing intensity in the latter stages of the exercise bout (3, 60, 61). This type of pace selection is referred to as parabolic pacing. Typically observed in exercise tasks of moderate-long durations (> 2 minutes), parabolic pacing is depicted by greater exercise intensities during the first and last 10 to 20 percent of an event (3), resulting in J, reverse J or U-shaped pacing profiles (**Figure 2.1d and 2.3**). The implication is athletes will access anaerobic metabolic pathways in the start and end sections of the trial to facilitate the higher exercise intensities. However, the anaerobic energy system is limited and less fatigue resistant due to producing metabolites associated with fatigue. For example, in 2000-m on-water rowing, Garland et al. (65) observed rowers will adopt a fast starting strategy, before mediating exercise intensity, to not comprise a fast finish or 'end spurt' in the later stages of the race. This approach is typical in endurance based events and may be influenced by a complex interplay between integrated feedback, both from internal and external stimuli, which build on prior knowledge and the anticipated duration of the exercise task (166). Further discussion of this observation to self-paced exercise will be explored in section 2.2.

Figure 2.3 Examples of J-shaped, reverse J-shaped and U-shaped pacing profiles. From Abbiss and Laursen (3).

Variable Pacing Strategies

Regular fluctuations to exercise intensity or work-rate is referred to as variable pacing (186). For many reasons, athletes will alter their pace throughout an exercise bout in response to both internal and external stimuli (3). External factors include the exercise duration, other competitors, type of feedback received or available and environmental factors (i.e. ambient temperature and humidity, oxygen availability, wind velocities and terrain) (40). In comparison, an athlete's internal physiological stimuli, goals and expectations before and during exercise are also considered essential determinants of athletic performance (40). In 2006, Tucker et al. (186) illustrated fluctuations in cycling PO are likely the result of complex interactions between incoming peripheral feedback and the anticipated exercise duration. The authors suggested that, where possible, future pacing studies should segment performance data into percentage 'bins' in such a way, where the resolution of the pacing profile is not compromised to allow inferences to pacing; therefore providing important clues about when athletes choose to up or downregulate the current work-rate (186). Employing a variable pacing strategy appears practical when exposed to variable winds or terrains (2). For instance,

performance may be improved when PO is increased on ascents and reduced when descending compared to trying to maintain a constant pace (170). Consequently, similar MPO's can still be maintained while counteracting physiological disturbances, where similar $\dot{V}O_2$, HR, blood lactate and RPE values have previously been reported between variable and even pacing strategies (98). However, if the magnitude of pace variation is too great (i.e. varied PO's within +5% of the mean trial PO or responding to changes in pace from a competitor), the current intensity may be intolerable leading to metabolic disturbances and premature fatigue (98). Nevertheless, studies examining the physiological implications of variable pace are inconclusive and further studies are required to better understand the effects and limitations of variable exercise intensities in response to external stressors.

Conclusion

A variety of pacing strategies are employed during exercise as athletes seek an acceptable strategy in response to various physiological and psychological stressors. If their strategy is successful, it is more likely to permit the best possible performance given the intrinsic and extrinsic constraints on the day. Understanding how and where athletes should best invest their energy is of great importance to coaches, sports scientists and researchers to optimise performance. As a result, many theories and models have been proposed in the research literature to describe the behaviour of exercise regulation which will be discussed in the following section.

2.2 THEORIES AND MODELS OF PACING AND EXERCISE REGULATION

Over the years, much debate has arisen around whether exercise intensity, and thus muscle activation, is regulated by the body's central or peripheral systems, resulting in several

models being proposed to explain this phenomena including: the teleoanticipatory theory (166, 190), the central governor model (130), the peripheral model (8), the perception based model (185), the psycho-biological model (111, 131) and the pacing awareness model (52). The viewpoints of these theoretical models will be discussed in the following section of this review.

2.2.1 Central versus peripheral models

In the 'central governor' hypothesis, exercise intensity is believed to pre-determined, built upon an athlete's knowledge of the exercise endpoint (130). Ulmer (190) first defined this concept as teleoanticipation, where it was proposed exercise performance may be regulated by a central control centre located somewhere in the central nervous system (CNS). In Ulmer's (190) model, it was suggested that athletes anticipate the work required to complete an exercise task, before work output is distributed accordingly. Noakes et al. (130) later proposed that the brain acts as a central control centre, which uses knowledge from prior experience and the anticipated duration as a reference point, to determine whether the current work-rate can be sustained in response to intrinsic (i.e. physiological, biochemical and cognitive) and extrinsic (i.e. environmental) sensory information. In turn, the authors proposed that not only does this 'central governor' preserve physiological homeostasis, by protecting against injury and damage to vital organs, it also ensures the chosen pace is within acceptable limits to prevent catastrophic performance failure (129, 130, 166). This concept was later expanded on by St Clair Gibson and colleagues (165) who suggested pace selection is an ongoing, continuous process during self-paced exercise, centred on complex interactions between incoming peripheral feedback and the anticipated exercise duration.

In accordance with the central controller hypothesis, numerous studies have demonstrated a relationship exists between exercise intensity (i.e. velocity and PO) and muscle activity (165). Differences in muscle activity, often reported as changes in integrated surface electromyography (iEMG), are attributed to changes in either the central drive in motor unit recruitment or the frequency rate of motor units at the measured working musculature (70). Therefore, a reduction in iEMG would be related to a decrease in central drive and marked decline in exercise intensity. In contrast, research has also demonstrated that iEMG may remain unchanged or increase despite decreases in PO, for example, during the final stages of a 4000-m cycling TT or 30-second cycle Wingate Test (79, 83).

Contrasting viewpoints between central and peripheral theories continue when attempting to explain exercise regulation where environmental factors have been manipulated. Specifically, it appears that the interaction of afferent sensory input to the CNS, and therefore, responding central motor drive, is delayed. For instance, earlier reports demonstrate cyclists inspiring hypoxic air (FiO_2: < 0.21) have implemented similar starting strategies to normoxic trials (normoxia FiO_2: 0.21), despite inspiring hypoxic air well before the beginning of the TT. Consequently, this observation supports the view that pace selection is pre-determined based on prior experience which is built on information available at the time and thus influences pre-task expectations (117). Therefore, prior knowledge and task expectations might mitigate the limitations of hypoxia in the early stages of the TT when athletes believe they were in normoxia (137, 190). However, as the exercise bout continued, cyclists did not appear to overcome the limitations of hypoxia, contrary to many models and theories of exercise regulation which emphasise the importance of prior experience overcoming peripheral feedback (117). St Clair Gibson and others (165) as well as Amann and colleagues (8) have therefore proposed the pacing phenomena it not simply a downregulation from central to

peripheral centres, instead they suggest it is an ongoing and complex interaction between the CNS (i.e. brain) and incoming peripheral feedback. For instance, blood chemoreceptors (137). group III-IV muscle afferents (10) or metabolic milieu (105, 126) are considered important regulators at the periphery to relay information to the CNS about whether the current exercise intensity is tolerable or not and whether changes are required.

Collectively, results to date suggest exercise regulation is not primarily determined by a central centre. Instead, a dynamic two-way "conversation" may occur (166), whereby, detection of disturbances in the muscle itself together with peripheral fatigue, may also provide vital information for the regulation of exercise and variations in pacing strategies (10). Therefore, the contribution of peripheral receptors (i.e. chemoreceptors of the carotid and aortic bodies in hypoxia) should also be considered when attempting to understand how physiological and psychological systems are maintained within certain critical limits during exercise (8-10). However, debate remains about the relative contributions and influence of central and peripheral centres relating to the modulation of exercise. Moreover, the contrasting opinions in the literature towards central and peripheral based theories argue these models primarily associate exercise performance to be regulated subconsciously, ignoring or disregarding conscious processes (52).

2.2.2 Perception based model

In 2009, Tucker (185) proposed a model suggesting athletes regulate exercise based on a template of subconscious, perceived effort. The model suggests if the level of effort is tolerable to reach the intended goals of a task, then the current work-rate is sustained as planned. However, if the athlete deems the current workload too great, they will adjust exercise intensity accordingly, based on prior experience, anticipation of the remaining

distance or duration, their estimation of their current capacities, motivation, biochemical disturbances at working musculature, environmental conditions and tactical decisions (185). The afferent pathway is sensitive to a number of stimuli, providing conscious awareness of essential subconscious regulatory processes. These include: pain, temperature, touch and muscle sensations (i.e. fatigue) (37). From a different perspective, Marcora's (112) psycho-biological model suggests perceived effort is consciously assessed moment-by-moment to prevent fatigue. A concept later explained by de Morree et al. (44) where perception of effort is the result of corollary discharge instead of afferent feedback from a central signaller. Despite disagreements of whether the experienced RPE is conscious (112) or not (185), both models agree the sense of effort strongly influences pace regulation.

In support of the perceived exertion template, Swart et al. (173) demonstrated that when cyclists were uncertain of the distance remaining in a 40-km TT, lower PO's and RPE were reported. Similarly, in a study by Mauger and colleagues (117) cyclists implemented a conservative approach during their first 4000-m TT, reporting lower PO's, $\dot{V}O_2$ and RPE which consistently increased as knowledge and familiarity with the exercise task developed over three subsequent TT's. However, Skorski et al. (160) revealed that RPE was consistent between 40-km cycling TT's regardless of the level of pre-accumulated fatigue. In addition, the authors reported lower HR, blood lactate and respiratory exchanges ratio responses in the fatigued state. The authors suggested this occurrence was either the integration of momentary RPE adapted central drive to ensure RPE followed a consistent template during the repeated exercise bouts, (185) or RPE is loosely associated with physiological variables and not the dominant 'driver' of events (111, 112). Recent work by Schallig and others (152) may further explain Skorski's findings; cyclists were instructed to complete a 10-km TT, however, at 7500-m cyclists were informed they had another 7500-m to complete. A transition phase

followed as the cyclists adjusted to this new information, demonstrated by significantly higher RPEs compared to a 15-km TT where the distance was known from the start and unchanged during the trial. Furthermore, the linear increase in perceived effort up to 7.5-km shifted to an almost horizontal line until matching RPE at a similar distance point during a known 15-km TT (~11.25-km) (152).

Collectively, these studies and theories provide further insight about where an athlete may make a decision to vary pace, or not, in relation to sensations of fatigue based around the information available to them at the time (185). If momentary perception of effort is too high and maintaining current intensity appears too great a risk, the pacing strategy may be moderated (52, 185). In contrast, if perception of effort is below what an athlete would normally expect at a particular point in the exercise bout, they might decide to increase their level of effort accordingly (52, 120, 185).

2.2.3 Decision making and pacing awareness models

The decision making model emphasises even in relatively simple exercise tasks, such as an individual TT, successful performance is dependent on the athlete's ability to correctly select and maintain an appropriate pacing strategy (42). To accomplish this, athletes must make a choice from available momentary information, where the consequences of their decisions are crucial to the performance outcome (69). These decision-making processes can be spontaneous or deliberate, made under periods of certainty or uncertainty, while evaluating the level of risk if acted on (69).

Before exercise commences decisions are made concerning how to implement an appropriate pacing strategy based on: performance goals and expectations (i.e. process or outcome),

previous experience, knowledge of current physical capacities, environmental (i.e. altitude, ambient temperature and humidity and wind conditions) and tactical considerations (i.e. other competitors, course profile and race tactics) (52, 146, 162). However, as the event continues, an athlete will receive additional information relating to performance: instantaneous performance measures or splits (i.e. time, velocity, PO or HR), changes, if any, to environmental conditions, physiological status received from incoming sensory feedback and responding to the behaviour of other competitors, all requiring the athlete to make a decision about whether to maintain or deviate away from the original strategy to achieve prior goals or expectations (67). Earlier work by St. Clair Gibson (165) proposed that the assessment of incoming sensory information creates a period of uncertainty about whether the current work-rate should or can be maintained, particularly if incoming signalling creates a mismatch with the pre-determined pacing template. Furthermore, the number of uncertain periods during events may increase the longer the exercise task (120), which therefore demands more decisions be made, often in the presence of fatigue. Consequently, cognitive function may be affected and potentially compromise accurate, informed decisions relating to whether the current level of effort is tolerable (112). For instance, if an athlete was uncertain in their ability to continue the current work-rate, when in fact they were capable, it may lead to an unnecessary reduction in exercise intensity, negatively impacting performance (17, 146). However, these periods of uncertainty have been shown to decrease with proximity to the finish line or exercise endpoint observed by an end spurt in the later stages of self-paced TT's (3, 17, 166).

More recently, to quantify these periods of 'uncertainty' or 'risk', De Koning et al. (43) developed an index derived from momentary RPE to predict the behaviour of individuals during simulated competition. The authors termed the index a Hazard Score. The Hazard

Score is calculated at segments (i.e. each kilometre) as RPE multiplied by the percentage of distance or duration remaining. Since its conception, a number of studies have shown athletes with a higher Hazard Score or perceived risk towards performance, are more likely to pace conservatively (59, 120, 174). However, Hutchinson and Tenenbaum (84) caution against using only a single-index measure, as RPE may be insufficient to capture and explain the range of sensations experienced during exercise, thus providing an oversimplification of psycho-physiological constructs. However, as Edwards and Polman (52) discuss, at what point information resides in different brain centres and moves across sub-conscious, pre-conscious and conscious states is difficult to discriminate. Therefore, despite several proposed theories attempting to explain decision-making processes during exercise regulation to date (52, 131, 146, 161), there is insufficient experimental evidence available to conclude on the manner in which decisions are made to select an appropriate pacing strategy.

2.2.4 Conclusion

Despite many models aiming to describe and understand the phenomena of pacing, the precise mechanisms of exercise regulation remain unclear. However, models do primarily agree pace selection is determined by an athlete's ability to resist fatigue. Furthermore, many models, acknowledge afferent sensory feedback from various physiological systems is received and regulated within the brain and integrated into the pacing strategy as an athlete responds to ongoing internal stimuli and external stimuli (70, 129, 130, 173). In addition, factors such as knowledge of task duration (152, 173) or current physical capacities (70, 117, 119, 129, 166), memory of prior experiences (117), motivation and mood (44) and tactical considerations (146), are also thought to be important factors in the regulation of exercise intensity. Collectively, further research is needed to understand the complexity of exercise regulation,

particularly surrounding central and peripheral fatigue as it occurs during a variety of exercise modalities, conditions and durations.

2.3 MANIPULATING EXERCISE REGULATION

In recent years, several meta-analyses have advanced our understanding of how environmental and task related feedback can affect overall performance, and whether these manipulations have a negative or positive effect on performance (45, 110). However, further studies are warranted to determine which part of the pacing strategy changes (i.e. start, middle and end) during exercise.

2.3.1 Environment manipulations on pacing

It is well documented various environmental conditions can influence athletes pace selection and performance. These factors include: oxygen availability (10, 34, 142, 187), heat-stress (134, 189), wind velocity (15, 172, 180) and environmental terrain (29, 172, 196). The purpose of this section is to compliment the meta-analysis in Chapter 3 by examining the implications of FiO_2, heat-stress and other environmental factors on pacing and performance during exercise.

Effect of manipulating oxygen availability during self-paced exercise

Endurance athletes rely on a high aerobic capacity, dependant on cardiac output and oxygen extraction (4, 47). Consequently, exercise performance at sea level (normoxia FiO_2: 0.21) is said to be limited by the availability of oxygen at the working musculature (46). Moreover, at rest, arterial oxygen saturation (S_aO_2) is ~ 100%, but can drop to < 90% during normoxic exercise (46, 48). Therefore, exercise above sea level (hypoxia FiO_2: < 0.21) poses a unique stress on physiological processes due to a lower partial pressure of oxygen which

progressively increases with elevation (45). As a consequence, athletic performance is impaired when inspiring hypoxia air (40).

Findings from a recent meta-analysis examining the effects of hypoxia on self-paced exercise reported TT performance was significantly impaired by ~16% (45). However, the authors reported a non-significant effect of hypoxia on exercise performance lasting less than two minutes in duration (45), supporting earlier work suggesting cyclists will initially adopt a similar starting strategy to normoxia during the early stages of a TT before pacing is adjusted (40). However, as exercise continues, the magnitude of impairment with acute hypoxia is dependent on the duration of exercise (45). For example, Deb et al. (45) demonstrated exercise between 2–10 minutes, and durations greater than 10 minutes further decreases exercise intensity by 18% and 27%, respectively. Deb et al. (45) recent meta-analysis supports previous experimental conclusions that a linear ~ 7% reduction in TT performance occurs per 1000-m of altitude gained, where TT's at higher altitudes may disrupt the pacing template in subsequent trials at lower altitudes (34). The disturbance to pacing is more pronounced for inexperienced compared to experienced participants (45). These findings are therefore supportive of previous theories that incoming sensory afferent feedback, knowledge of the trial distance or duration (173), current momentary perceived effort influenced by prior experience (117, 152), current expectations (such as outcome and strategic goals) (19) and knowledge of current physical capacities (146, 161) are integrated into decision-making and regulatory processes during self-paced exercise.

It is well established that increasing the FiO_2 during endurance exercise permits a greater exercise intensity or capacity to be achieved (10, 146). To accomplish this, researchers have administered hyperoxic air (FiO_2: > 0.21), where athletes inspire supplemented oxygen during fixed and self-paced exercise (10, 11). In a recent meta-analysis exploring the effects of self-

paced exercise in hyperoxia, nine different intervention studies were identified (110). It was found athletes who inspired hyperoxic air (FiO_2: 0.33 to 1.0) improved self-paced performance by ~ 6% (110). For instance, in rowing, Peltonen et al. (135) found that national level male rowers inspiring hyperoxic air (FiO_2: 0.62) improved performance time by ~ 11.5 seconds during a 2500-m rowing ergometer TT compared to normoxia. The observed improvement in hyperoxia was associated with ~ 6.5% increase in mean power outputs (MPO), concomitant to an increased $\dot{V}O_2$ after 500-m compared to the normoxic TT (135). In contrast, Amann and colleagues (10) administered higher oxygen content (FiO_2: 1.0) and reported ~ 13.6% improvements in PO compared to a normoxic 5-km TT, where normoxic, hypoxic and hyperoxic TT's were performed in a randomised fashion. Nevertheless, moderate levels of hyperoxia (FiO_2: 0.30) induce similar improvements in TT performance as those performed at more extreme increases in FiO_2 (126, 135, 187, 198). At this point, however, previous studies have blinded athletes to the FiO_2 (40, 110). Therefore, whether knowledge of inspiring hyperoxia encourages a decision to upregulate exercise intensity or not, compared to a normoxic trial, remains largely unknown. Nevertheless, it is clear inspiring supplemented oxygen, compared to normoxia results in a greater total volume of work accomplished (110). However, Tucker et al. (187) suggest the rate of development of peripheral muscle fatigue appears to be similar compared to normoxia, where at a given time point, similar levels of muscle activation are required to produce the same PO. This finding is in agreement with earlier reports, where athletes will adjust exercise intensity despite being unaware of inspiring hyperoxic air, suggested the result of associated mechanisms responding to incoming sensory feedback from the periphery (10, 87, 137).

Improved performances in hyperoxia have previously been attributed to an increase $\dot{V}O_2$ and change in $\dot{V}O_2$ kinetics (137), consequently increasing oxygen availability at skeletal muscles

and reducing biochemical and physiological disturbances to homeostasis (187). For instance, inspiring supplemented oxygen increases cerebral oxygenation (126) and reduces levels of metabolic by-products (i.e. lactate, hydrogen ions, inorganic phosphate) at the working musculature (105), often limiting factors for exercise performance in normoxia (187). It is important to note, accumulation of fatiguing metabolites still occurs during hyperoxic exercise bouts, however, critical levels are reached later or at higher exercise intensities compared to normoxia (110). In addition, other factors, such as the effectiveness of respiratory muscles to uptake oxygen, muscle recruitment and teleoanticipation have also been considered important influences on exercise intensity in hyperoxia before limiting levels of fatigue are reached (10, 137, 187). Collectively, improved performance in hyperoxia is thought to be facilitated by an increased aerobic metabolism which is compromised in hypoxia (135-138).

Effect of heat-stress on self-paced exercise

A large quantity of energy is dissipated as heat when performing exercise (86). Consequently, exercise in environments where the ambient temperature, humidity or both are high, imposes additional external stress on athletes (86). Furthermore, exercise in hot conditions can become more problematic in continuous exercise lasting over 30 minutes in duration, as the gradient for heat exchange between the skin and environment and evaporative heat loss is reduced. As a result, core body temperature rises and performance is impaired (86).

Based on previous research, it appears that in hot conditions athletes will initially adopt a similar pacing pattern to temperate conditions for exercise 10 to 15 minutes in duration, before exercise intensity is reduced to avoid excessive rises in core temperature (40). This observation appears consistent across a range of different exercise modalities, including: cycling (53, 141, 175, 189), running (53, 155, 168) and triathlon (167). However, previous

reports have demonstrated that if participants are inexperienced at TT's in heat, they may misjudge the start section of the TT by beginning the trial too aggressively (31, 153). Once exercise continues, it is thought factors such as significant increases in skin temperature (86), perceived exertion (38), and thermal sensation (153) are potential afferent drivers of the adjustment in the pacing strategy, which is reduced in the middle and later stages of an exercise in heat compared to temperate conditions (40).

Current understandings underpinning exercise regulation in heat are fourfold: i) homeostatic processes redirect blood flow to dissipate heat at the skin (80); ii) this becomes a concern in long duration exercise due to compromised blood flow to the working musculature, as a result, a reduction in gross mechanical efficiency occurs (80); iii) if differences occur between the rates of metabolic heat production and net heat loss, and the exchange of evaporative heat, core temperature will increase (86); iv) to compensate for this, maintenance of blood pressure takes priority over blood flow to the skin and working musculature and an excessive heat-stress will occur (30).

It is clear exercise in heat-stress has detrimental effects on overall performance. However, despite what is currently known, the probability that a difference in pacing strategy is practically meaningful across various ambient temperatures and durations remains unclear.

Influence of other environmental factors on self-paced exercise

In general, athletic training and competitive endurance events take place outdoors. Therefore, in addition to oxygen availability, heat and humidity, other environmental factors such as wind velocity (15, 172, 180) and environmental terrain (i.e. gradients) (29, 172, 196) have previously been shown to influence pacing and performance. For example, in mass start cycling races, team tactics relating to course gradients or profile and drafting techniques to

mitigate wind conditions are important considerations for the constraints on the day of performance. Best performances are often achieved on flat courses with no wind as constant PO's are more easily maintained, minimising fluctuations in pacing and metabolic disturbances (61). However, over hilly courses, variable pacing occurs as athletes adjust to travelling up or down different gradients and may implement tactics to gain a competitive advantage (61). For instance, group or individual breakaways on ascents or aggressive downhill descending have been observed in major cycling competitions. In 1997, Swain et al. (172) using a mathematical model over a hilly course profile observed the implications of even and variable pacing strategies over a 10-km cycling bout. Despite both approaches reporting similar MPO's, variable pacing showed improvements in performance time if cyclists were to increase PO on ascents and reduce PO when descending compared to trying to maintain a constant pace. Although research in this area is limited, a small number of research studies provide support of Swain's model (172). For example, Cangley et al. (29) showed variable pacing strategies over a 4-km undulating course can improve cycling performance time by 2.9% compared to maintaining constant PO's. The authors suggested the tenth placed rider in the 2008 UK National 16.1km TT championship would have placed third if they had implemented a variable pacing strategy (29). Moreover, Atkinson and Brunskill (15) demonstrated that fluctuations in pace during 16.1-km cycling TT's in head and tailwinds was a viable tactic to minimise the effects of wind resistance and accumulated rate to metabolic disturbance. It was found compared to holding an even pacing strategy, time to completion was improved when PO was increased by 5% into a headwind and reduced by 5% into tailwinds (14).

Conclusion

Extensive research assessing regulation in different exercise environments provides further insight into existing theoretical models. It seems apparent that manipulating any form of

environmental factor has considerable effect on task performance. Therefore, further research on the effects underpinning performance will assist our understanding of how athletes should appropriately pace in differing environments.

2.3.2 Feedback manipulations on pacing

It is well established that manipulating incoming information available to athletes can influence pace selection and performance (3). To achieve this, researchers may withhold or deliberately misinform athletes of the true purpose and procedures of the study (90, 204). Examples include: unknown, incorrect or unexpected changes to exercise duration; time deception; psychological manipulations (i.e. altering self-efficacy towards the exercise task); inaccurate feedback relating to performance metrics; and the presence of a competitor (**Figure 2.4**) (90, 204). However, the effectiveness of deceptive methods appears to be largely dependent on the type of variable manipulated, discussed in greater detail in Chapter 3 (40). Despite alterations to pacing strategies, deception of time, distance, and HR have been shown to be less effective in eliciting performance improvements (204). Furthermore, a limitation for several studies is the exercise intensity during deception TT's has been fixed (18, 24, 35, 55). As a result, studies investigating the effects of deception during maximal exercise are limited (204).

In a recent review, the non-invasive provision of deceptive information, regarding exercise performance or the task environment, was suggested the most successful form of external task manipulation (40). This type of deceptive feedback, known as performance deception, is achieved by altering task feedback such that athletes believe they are performing better (positive) or worse (negative) than reality, where performance metrics such as velocity, PO, elapsed distance or time have been manipulated (40, 90, 204). Therefore, the following

sections aim to compliment the meta-analysis in Chapter 3 by providing further insight of how performance deception influences the regulation of maximal exercise, and additionally, how these manipulations influence subsequent trials after deception is revealed.

Figure 2.4 The possible interventions that can modify exercise performance adapted from Noakes (128).

Positive deceptive feedback

Informing athletes they are performing better than reality, defined as positive deceptive feedback, is typically achieved by informing athletes they have completed more distance (6, 56, 117, 132, 173, 197, 206); are maintaining greater exercise intensities during stages of a TT (75, 119, 176); or by misinforming them of ambient and core body temperatures (31). In two 20-km cycling TT's, Micklewright and colleagues (119) examined the effects of informing cyclists they were maintaining velocities 5% above actual values, compared to a final TT where accurate feedback was provided. Overall, no significant changes were found for performance time, speed or PO. However, participants did adopt a more aggressive starting strategy during the first 5-km of the positive deception TT (119). Similarly, Taylor et al. (176) found no performance improvement when informing triathletes their running velocities were

3% above a baseline trial. However, a non-significant trend for the fastest run time was reported for the deception trial compared to baseline during a 1.66-km treadmill run. In addition, Puleo and Abraham (144) reported no performance improvement occurred during 30 minute treadmill running, when untrained runners were informed their split times were 5% faster than their actual pace at five minute intervals. Similarly, informing participants they had completed more distance than in reality during cycling TT's appears to be ineffective in changing pacing to enhance performance (56, 127, 132, 206). A result explained by Paterson and Marino (132) is likely due to TT performance being regulated based on perceived effort rather than the informed moment-to-moment distance feedback. However, if TT distance or duration is unknown or changed during performance it may increase the level of uncertainty surrounding the exercise bout and negatively impact performance (117, 165). Collectively, it would appear athletes receiving positive deceptive feedback, relating to velocity, distance or time make little or no change to pace regulation, supporting the perceptual 'template' theory proposed by Tucker (185) which is considered to be robust in self-paced TT's. More specifically, it appears afferent feedback mediates perceived exertion from incoming sensory information based on pre-exercise goals and expectations and information available at the time (52, 75).

Another approach in positive deception feedback studies has been to misinform athletes of physiological variables to examine the management of effort across the duration of an exercise bout. Recently, Gordon and colleagues (72) demonstrated that when untrained runners were informed their HR was 10 beats per minute above actual values, this influenced the manifestation of the $\dot{V}O_2$ plateau, such that pacing strategy was disrupted. The authors concluded non-contingent feedback, in the form of HR, may promote the sparing of the finite

anaerobic capacity to compensate for the mismatch between afferent signal and perception of effort.

In a different approach, Castle et al. (31) informed cyclists the ambient (6°C) and their core body temperatures (0.3°C) were lower or higher than reality during 30 minutes of ergometer cycling. As a result, cyclists in this experiment were able to mitigate the limitations of ambient temperature when believing it was below real values, suggesting belief may have influenced performance, where a slight upregulation in MPO was found compared to believing the ambient and core temperatures were hotter than reality (75). However, this was also associated with a greater variability in performance. A recent meta-analysis provided a potential explanation for this occurrence suggesting a change in ambient temperature may be easier to detect from the periphery (40), due to thermal sensations and comfort (153, 156), potentially creating a mismatch between actual and anticipated levels of effort and exertion with the known temperature revealing deception (85, 106, 176).

Negative deceptive feedback

At present, altering task feedback where athletes believe they are performing worse (negative feedback) than reality, appears to be the most effective method to elicit performance improvements, both during deception trials themselves, and in a subsequent trial after deception is revealed (40). Negative deception is typically achieved via misrepresentation of a previous performance trial in the form of an on-screen avatar, which is programmed above previous best performances (i.e. 2% greater) unknown to the athlete (40, 89, 91, 158, 169, 203). In comparison, research attempting to disturb the pre-determined pacing template by informing participants they had travelled less distance than in reality has been equivocal (18, 19, 35, 55, 56, 127, 206).

Performance improvements associated with negative deception trials are believed to be the result of athletes accessing 'reserve capacities', considered the remaining metabolic energy after a self-paced task, maintained presumably to prevent catastrophic failure of homeostasis (173). Previous work by Stone et al. (169) attributed this to a greater anaerobic energy contribution at 90% of the completed distance during a 4000-m TT, associated with significant improvements in MPO. Similarly, work by Jones and colleagues (89, 91) demonstrated during cycling TT's of the same distance, racing against a pacer programmed 2% above baseline velocities, (unbeknownst to cyclists), produced a greater respiratory exchange ratio, indicating negative deception elicited a greater anaerobic energy contribution across the trial. Performance improvements appear not to alter the pacing template, instead negative deception facilitates a subtle upward shift in exercise intensity compared to a baseline trial (158). However, further evidence has revealed MPO manipulated 5% above baseline appears to not result in performance improvement, instead causing early peripheral disturbance leading to premature fatigue, despite athletes reporting high levels of motivation levels before TT's (171). Therefore, it appears an upper limit may exist to how much deception can be tolerated.

Although the meta-analysis outlined in Chapter 3 supports the provision of negative deceptive feedback to improve performance in cycling, limited research has been performed in other exercise modalities (204). An exception is a recent study by Taylor and colleagues (177) who reported similar findings to our meta-analysis during the cycle leg of a sprint-distance simulated triathlon. In their research, when non-elite triathletes were deceived of cycling MPO by 5% above a baseline trial, significant improvements in performance time were found. However, the manipulated MPO increase in this study was not represented by an on-screen pacer as implemented in earlier investigations (89, 91, 158, 169, 171). Instead,

instantaneous PO and MPO was displayed on-screen via a large television screen paired with the cycling ergometer where participants were required to maintain the prescribed PO for the entirely of the 500 kJ cycling bout (177). Performance improvements were associated with elevated HR and blood lactate concentrations, while RPE was unchanged in the deception trial compared to baseline. Therefore, in support of with previous work (17, 146), these findings further suggest the subtle upregulation in exercise intensity from negative deception feedback is manageable, leading to improved performance. However, despite recent advancements in this area, the upper limit to reserve capacities is largely unknown. Consequently, further evidence for techniques to exploit reserve capacities during competitive events requires further research.

Exercise performance after deception is revealed

A major focus of performance deception feedback is an attempt to deviate athletes away from their routine pacing strategy, and therefore, examine whether performance improvements can be sustained in a subsequent trial after deception is revealed. To date, deception interventions of this nature show athletes can retain performance gains after deception is revealed up to 2% above their previous best baseline performance (158, 169, 171). Possible mechanisms explaining these improvements are thought to be an upward shift in PO without raising RPE where the exercise intensity may become intolerable (89, 91, 158). Here, the subtle increase in work-rate appears manageable despite athletes reporting higher levels of perceived exertion (158). In comparison, it has been suggested the performance improvement after negative deceptive cycling TT's, may be explained by the presence or absence of a pacer as opposed to altered performance beliefs, irrespective of whether cyclists were aware of deception in a previous trial or not (89). However, deception studies by Jones et al. (89) manipulated speed by +2% before an informed TT, compared to increasing MPO by +2% in other investigations (158, 169). Consequently, the disparity of opinions in exercise performance after deception is

revealed before a subsequent TT, may be explained partly by the non-linear relationship between speed and PO, where models indicate a 1% increase in speed equates to a ~ 2.9% increase in MPO (57). Collectively, the use of deception appears to be a useful tool in understanding how athletes pace. However, further investigation to advance our understanding of how deception may improve subsequent performances after it is revealed, and whether deception may become a viable training tool warrants further exploration.

Influence of a competitor

An added variable in deception interventions is the addition of an on-screen avatar which takes the form of a competitor. Therefore, if unaccounted for, the presence of an avatar could be perceived as either competition or motivational support (89, 157, 203, 205). For example, previous work by Konings et al. (94) has shown during 4000-m cycling TT's, cyclists will upregulate MPO, improving performance time, when racing an on-screen avatar accurately representing the MPO from their previous performance. However, RPE did not differ between competitor and no competitor TT's. A further study by Konings and colleagues (95) revealed the behaviour of the pacer, where the virtual opponent started slower and finished faster or the pacer started faster and finished slower, may also affect pacing. Specifically, a faster starting opponent encourages greater MPO by cyclists in the initial 750-m compared to a slower starting opponent during 4000-m TT's. Nevertheless, performance times were identical compared to a self-paced TT irrespective of the pacer behaviour.

In agreement with earlier opinions in rowing (36), Jones and colleagues (89) suggested the presence of an on-screen pacer provides additional motivation and might partly explain the increases in PO observed in performance deception TT's (158, 169). In contrast, Bath et al. (22) reported that although athletes perceived their performance had improved, the addition of a second runner who ran at an equal, slower or faster pace to participants did not significantly

alter pace selection, velocities, HR or RPE across five 5000-m TT's. These findings led the authors to conclude an athlete's pacing template is robust and pre-determined before the event (22). However, in Bath et al. (22) investigation, the second runner was never introduced as an opponent, a factor which may partly explain the disparity to later investigations (158, 169). Nevertheless, findings by Bath et al. (22) were supported in a recent meta-analysis, where the presence of a 'competitor' pacer, accurately representing MPO from a previous TT, did not demonstrate a significant improvement in trial MPO compared to a baseline (control) TT where no pacer was present (40).

To date, studies investigating the influence of a competitor have been equivocal and likely the result of different methodological approaches. Nevertheless, the performance advantage, or not, of a competitor should be considered and accounted for within future study designs. However, at present, how athletes perceive a virtual or real world competitor remains largely unknown, warranting further investigation to advance our understanding of how competition is perceived.

2.4 CONCLUSION

The present supplementary literature review has examined i) the significance of pacing and why it is important to understand what is occurring within athletic performance itself, ii) the various models used to understand the pacing phenomena in self-paced exercise, iii) self-paced observations in different environmental conditions, and iv) common techniques by which to deceive athletes during exercise. To date, there is a lack of definitive research investigating how knowledge of inspiring supplemented oxygen alters self-paced exercise, and specifically, whether acute exposure can improve subsequent performances in normoxia. This is surprising given the known ergogenic effects of inspiring supplemental oxygen and the availability of using hyperoxia as a training modality. Furthermore, whether a performance reserve exists in hyperoxia and the mechanisms underpinning exercise regulation requires further exploration. At this point, it also remains unclear how oxygen and negative performance deception, may change task expectations to alter pacing such that performance in normoxia is improved. Collectively, this information will further our understanding of how hyperoxia and deceptive information alters trained athletes' exercise regulation and further, determine if these techniques could be used in training to enhance performance. This thesis therefore aims to examine how environmental and performance feedback manipulations may affect exercise regulation and subsequent performances in trained cyclists.

3.0 STUDY ONE: THE EFFECT OF ENVIRONMENTAL AND FEEDBACK INTERVENTIONS ON PACING PROFILE IN CYCLING: A META-ANALYSIS.

Citation: **Davies, M. J**., Clark, B., Welvaert, M., Skorski, S., Garvican-Lewis, L. A., Saunders, P., & Thompson, K. G. (2016). Effect of environmental and feedback interventions on pacing profiles in cycling: a meta-analysis. *Frontiers in physiology*, *7*, 591. [Appendix 1]. [https://doi.org/10.3389/fphys.2016.00591]

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3.i FORM E: DECLARATION OF CO-AUTHORED PUBLICATION CHAPTER

For use in theses which include publications. This declaration must be completed for each co-authored publication and to be placed at the start of the thesis chapter in which the publication appears.

Declaration for Thesis Chapter 3

Declaration by candidate

In the case of Chapter 3 the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Study design, data collection and analysis and study write up	70

The following co-authors contributed to the work.

Name	**Nature of contribution**	**Extent of contribution (%)**	**Contributor is also a student at UC Y/N**
Bradley Clark	Assistance with study design, interpretation of results and proof reading of drafts	7	N
Marijke Welvaert	Assistance with study design, analysis and interpretation of results and proof reading of drafts	5	N
Sabrina Skorski	Assistance with study design, data collection, analysis and interpretation of results and proof reading of drafts	5	N
Laura Garvican-Lewis	Assistance with study design, interpretation of results and proof reading of drafts	3	N
Philo Saunders	Assistance with study design, interpretation of results and proof reading of drafts	3	N
Kevin Thompson	Assistance with study design, interpretation of results and proof reading of drafts	7	N

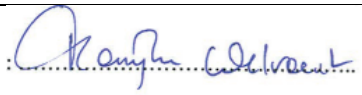
Candidate's Signature		**Date** 24/04/2018

Declaration by co-authors
The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors.
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s)	Australian Institute of Sport Department of Physiology, Bruce, ACT Research Institute for Sport and Exercise, University of Canberra, Bruce ACT

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant.]

Co-author Signature Number	**Co-author's Signatures**	**Date**
Signature 1		24/04/2018
Signature 2		24/04/2018
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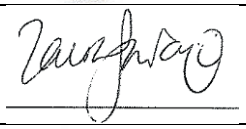
Signature 4		24/04/2018
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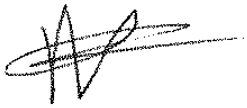
Signature 6		24/04/2018

3.1 ABSTRACT

Background: To date, a meta-analysis investigating the effect of different environmental and extrinsic manipulations on the pacing strategy of participants during self-paced time-trials is not available. The aim of this study was to address this shortfall.

Method: The PubMed, SPORTDiscus and MEDLINE (via EBSCO) databases, including citation tracking of key review articles were searched up to September 2016. 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*.

Results: 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 manipulated pre-cooling interventions and seven studies manipulated various forms of feedback. Mean power output (MPO) was observed to significantly reduce 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, hyperoxia and pre-cooling strategy conditions did not demonstrate a significant change in trial or section MPO 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 ($p > 0.05$).

Conclusion: The available data suggests exercise regulation in hypoxia and heat-stress is modified 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, endurance performance, hypoxia, deception, hyperoxia, heat-stress, pre-cooling, feedback

3.2 INTRODUCTION

The ability to appropriately distribute energy expenditure throughout an exercise task is critical in order to optimise athletic performance (3, 70). 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 speed) during athletic competitions in which athletes are free to vary their exercise intensity (44, 160). 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 (3, 70). Several models have been proposed to explain this phenomena including: the teleoanticipatory theory (166, 190), the central governor model (130), the perception based model (185), the pacing awareness model (52) and the psychobiological model (111, 131). These models generally 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 (70, 111, 129, 130). In addition, factors such as knowledge of the task duration or distance remaining (173), memory of prior experiences (117) and motivation and mood (44) 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 (188). These factors include oxygen availability (10, 34, 142, 187), heat-stress (134), wind velocity (180), hydration status(50), carbohydrate (5) and caffeine ingestion (201), pre-cooling strategies (49), motivation (82), fatigue (160), deception (91, 158, 169), pacing feedback (183, 184) and music (16). 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 (90, 204), decision making (52, 118, 146, 161) and neurophysiological determinates (147) 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.

3.3 METHODS

A computerised literature search was undertaken, using 18 different keywords (*'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 Preferred Reporting Items for Systemic Reviews and Meta-Analyses (PRIMSA) checklist (23, 103) and the search strategy proposed by (81). Combinations of these words were used to systematically search 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.

3.3.1 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 3.1**. 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 (randomised 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 (10, 28, 175), to normalise 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 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 written in English and published in an internationally peer-reviewed scientific journal.

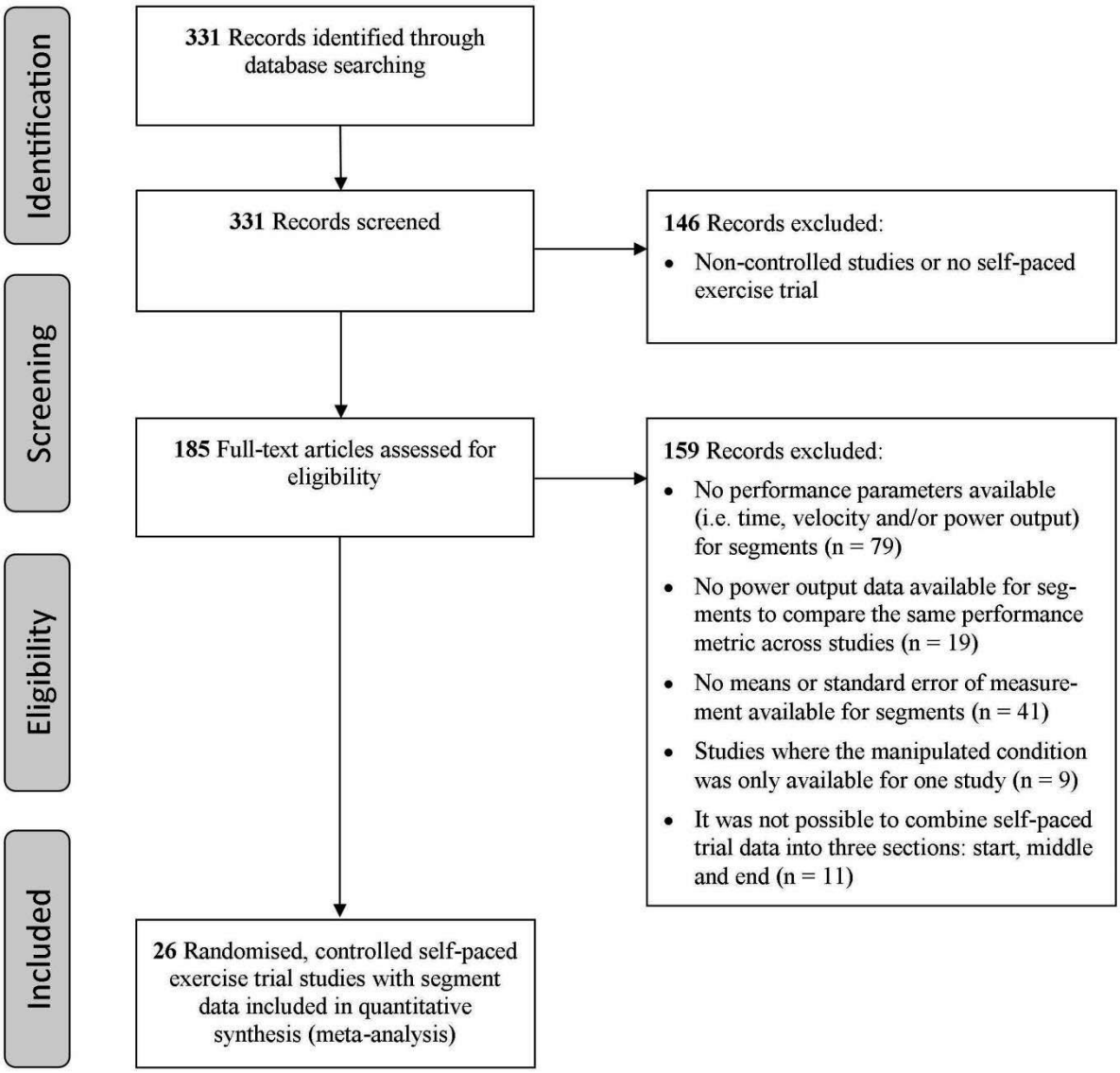


Figure 3.1 Flow chart summary of the study selection process.

3.3.2 Classification of the studies

Of the initial 185 peer-reviewed studies identified, a total of 26 studies satisfying the inclusion criteria were analysed. 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 (FiO_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 (e.g. hypoxia, FiO_2: < 0.21; normoxia FiO_2: ~ 0.21; iso-oxia, FiO_2: > 0.21 to 0.30; hyperoxia, FiO_2: > 0.30), 2) heat-stress (> 23°C, relative humidity < 70%), 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 travelled a greater distance than they actually had or informed that the ambient and their body 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 were deceived based on a previous performance (e.g. MPO was increased compared to the previous (baseline) trial but the participants were unaware); and finally informed deception, where participants were informed they had been deceived in a previous trial before completing a final trial with a known MPO increase in the presence of a pacer (158) or not (89, 91).

3.3.3 Data extraction

Due to the various trial durations examined in the existing literature, whole trial MPO and segment power output for all studies was extracted from control and intervention conditions,

allowing performances to be segmented into start, middle and end sections (i.e. into thirds) for further comparison. A large proportion of the analysed 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 millimetre. 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 (99). 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 (99).

3.3.4 Study quality assessment

Although it was not a requirement for the inclusion criteria, the Physiotherapy Evidence Database assessment criteria of study quality (PEDro) scale (109) 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 0–11. A score ≥ 7 is considered 'high quality', a score of 5 or 6 is deemed 'moderate quality', and ≤ 4 defined as 'poor quality' (109). In addition, qualitative participant-group descriptions based on performance levels on a scale of one to five was defined for each study as previously described (133). Briefly, participant-groups were referred to as performance level one when studies described their participants as '*untrained' or 'sedentary'*; terms

'healthy, 'habitually active', 'physically fit', 'physically active', 'active', and *'habitually physically active'* were considered recreationally trained and referred to as performance level two; trained participant-groups defined as '*trained', 'trained competitive', 'endurance-trained',* and '*competitive'*. were classified as performance level three; level four contained terms *'highly-trained', 'well-trained', 'well-trained competitive', 'highly-trained competitive',* and *'national cycling league'*; and level five included studies defining their participants as *'professional cyclists'* (133).

3.3.5 Data analysis

The meta-analysis was performed in statistical Software R (Vienna, Austria: R Foundation for Statistical Computing) using the package metaphor (195). The mean difference (MD) between control and intervention trial for each study was analysed 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% 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) (101, 208) 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:

*(Segment mean power output / 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 was analysed using an exploratory graphical analysis of the difference between control and experimental condition IP (i.e. control IP – experimental IP for each individual segment). This was termed the Pacing Index (or IP) Change Score.

3.4 RESULTS

3.4.1 Search results

In total 331 articles published in English were found, out of which 185 were identified as peer-reviewed controlled studies. These articles were evaluated according to the specified inclusion criteria (**Figure 3.1**) and we identified 26 studies with a total number of 351 subjects that met all inclusion criteria. Almost all studies used a randomised cross-over design, except for studies (89, 91, 155, 162, 202) 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 (31) analysed the effects of heat-stress or deceiving participants of ambient and body temperature by providing positive deceptive feedback, thus data were included in two themes, heat-stress and feedback, respectively. Participant's maximal oxygen uptake ($\dot{V}O_{2max}$) from the 19 studies which reported this parameter was (mean ± SD) 61.3 ± 4.6 $mL \cdot kg^{-1} \cdot min^{-1}$. Three studies only provided peak power output (PPO) data (393 ± 51 W), while four studies did not disclose participant's $\dot{V}O_{2max}$ or PPO. Most studies exclusively investigated male participants, except for one, which reported having one female participant amongst a male

cohort (139). However, one study did not report the sex of their participants (155). An overview of included studies is provided in **Table 3.1**.

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, however in other conditions it is possible to blind participants to the intervention condition and in 11 studies this was the case. In these studies participants were blinded to a manipulation in: oxygen availability (n = 4) (10, 34, 142, 187), pre-cooling (n = 1) (20) and feedback (n = 6) (6, 31, 89, 91, 158, 197). Only one study implemented a double-blind design (34). **Figure 3.2** illustrates the number of research trials that fulfilled each criteria of the PEDro scale.

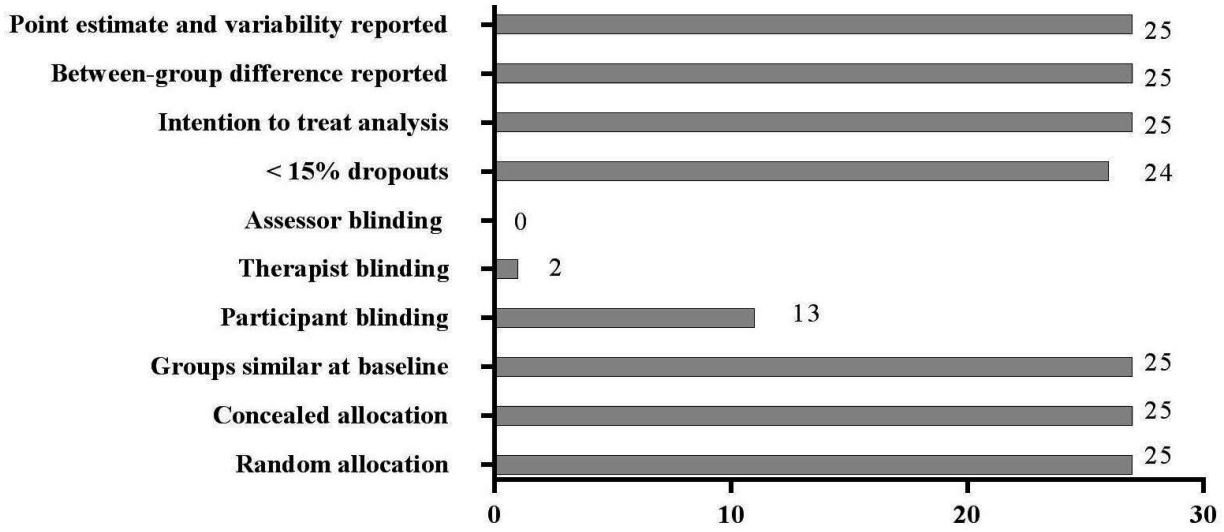


Figure 3.2 Number of studies meeting individual PEDro [Physiotherapy Evidence Database] criteria.

Table 3.1 Overview of the analysed studies

Study, year	Study Design	No. of participants and training status[a], Participant Classification Score	Exercise protocol	Intervention	Time point of environmental exposure or intervention manipulation	Performance measurement (total time (s or min) or mean power output (MPO) in watts)	PEDro Score
OXYGEN AVAILABILITY							
Amann et al. (10)	Crossover	8 Well-trained male cyclists (63.3 ± 1.3 ml $kg^{-1}min^{-1}$), Level 4	5 km cycling TT	TT 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 = 7.3 ± 0.1 min FiO_2: 0.15 = 8.1 ± 0.1 min FiO_2: 0.24-0.30 = 5.9 ± 0.1 min FiO_2: 1.00 = 7.3 ± 0.1 min	9
Clark et al. (34)	Crossover	10 Well-trained male cyclists/ triathletes (67.7 ± 1.3 ml $kg^{-1}min^{-1}$), Level 4	5 min cycling TT	TT in normoxia (FiO_2 = 0.21; CON), hypoxia (FiO_2 = 0.19; 0.16; 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
Periard and Racinais (142)	Crossover	12 Well-trained male cyclists, Level 4	Cycling TT to 750 kJ	TT 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. (187)	Crossover	11 Well-trained male cyclists (395 ± 33 PPO), Level 4	20 km cycling TT	TT 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. (5)	Crossover	10 Well-trained male cyclists (61.7 ± 5.0 ml $kg^{-1}min^{-1}$), Level 4	16.1 km cycling TT	TT in temperate (18.1ºC, 58% RH; CON) and heat (32ºC, 50% RH; INTER)	Exposure during warm-up (90 min fixed workload) and at rest. Total of 116.3 min in heat before trial	18.1ºC = 25.4 ± 1.6 min 32ºC = 27.5 ± 1.9 min	8

Table 3.1 Continued

Study, year	Study Design	No. of participants and training status[a], Participant Classification Score	Exercise protocol	Intervention	Time point of environmental exposure or intervention manipulation	Performance measurement (total time or mean power output (MPO) in watts)	PEDro Score
Altareki et al. (7)	Crossover	9 Competitive male triathletes and cyclists (61.7 ± 8.6 ml $kg^{-1}min^{-1}$), Level 3	4 km cycling TT	TT in cool (13ºC, 40%; CON) and heat (35ºC, 60% RH; INTER)	Exposure before, during and after warm-up. Total of 21 min in heat before trial	13ºC = 6.4 ± 0.3 min 35ºC = 6.5 ± 0.3 min	8
Castle et al. (31)	Crossover	7 Recreational male cyclists (58.8 ± 5.7 ml $kg^{-1}min^{-1}$), Level 2	30 min cycling TT	Time-trial in temperate (22ºC, 43% RH; CON) and heat (31ºC, 64% RH; INTER)	Exposure during a 7 min warm-up in heat before trial	22ºC = 179.9 ± 50.9 MPO 31ºC = 168.1 ± 54.1 MPO	9
Peiffer and Abbiss (134)	Crossover	9 Trained male cyclists (60.5 ± 4.5 ml $kg^{-1}min^{-1}$), Level 3	40 km cycling TT	TT in temperate (22ºC, 40% RH; CON), cool (17ºC, 40% RH; INTER), warm (27ºC 40% RH; INTER), and hot (32ºC, 40% RH; INTER)	Time point unknown, including temperature in warm-up	17ºC = 58.8 ± 2.0 min 22ºC = 59.0 ± 2.3 min 27ºC = 59.1 ± 2.3 min 32ºC = 60.7 ± 2.9 min	8
Périard et al. (141)	Crossover	8 Well-trained male cyclists (66.4 ± 5.3 ml $kg^{-1}min^{-1}$), Level 4	40 km cycling TT	TT in temperate (20ºC, 40%: CON) and heat (35ºC, 60% RH; INTER)	Exposure 5 min before trial, after a warm-up in temperate	20ºC = 59.8 ± 2.6 min 35ºC = 64.3 ± 2.8 min	8
Périard and Racinais (139)	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	TT in temperate (20ºC, 40%; CON) and heat (35ºC, 60% RH; INTER)	Exposure 5 min before trial, after a warm-up in temperate	20ºC = 48.8 ± 12.7 min 35ºC = 55.8 ± 14.4 min	8
Periard and Racinais (142)	Crossover	12 Well-trained male cyclists, Level 4	Cycling time-trial to 750 kJ	Time-trial in temperate (18ºC, 40%; CON) and heat (35ºC, 60% RH; INTER)	Exposure 5 min before trial, after a warm-up in temperate	18ºC = 48.2 ± 5.7 min 35ºC = 55.4 ± 5.0 min	8

Study, year	Study Design	No. of participants and training status[a], Participant Classification Score	Exercise protocol	Intervention	Time point of environmental exposure or intervention manipulation	Performance measurement (total time or mean power output (MPO) in watts)	PEDro Score
Schmit et al. (155)	Parallel group	34 Well-trained triathletes, sex unknown. Temperate (n = 22; 63.3 ± 2.1 ml $kg^{-1}min^{-1}$), Heat (n = 12; 62.2 ± 3.6), Level 4	20 km cycling TT	TT in temperate (21ºC, 50%; CON) and acute heat exposure (35ºC, 50% RH at 0; INTER and 11 ± 4 days; INTER)	Exposure 15 min before trial during a warm-up, the second heat trial took place 11 ± 4 days after trial 1 with no acclimatisation	*0 days* 21ºC = 32.2 ± 2.0 min 35ºC = 33.2 ± 1.58 min *11 ± 4 days after first trial* 21ºC = 31.5 ± 1.4 min 35ºC = 32.4 ± 1.2 min	7
Tatterson et al. (175)	Crossover	11 National male road cyclists (66.7 ± 13.6), Level 5	30 min cycling TT	TT in temperate (23ºC, 60% RH; CON) and heat (32ºC, 60% RH; INTER)	Time point unknown, including temperature in warm-up	23ºC = 345 ± 9 MPO 32ºC = 323 ± 8 MPO	8
Tucker et al. (189)	Crossover	10 Recreational male cyclists (376 ± 47 PPO), Level 2	20 km cycling TT	TT in cool (15ºC, 60% RH; CON) and heat (35ºC, 60% RH; INTER)	Time point unknown, including temperature in warm-up	15ºC = 28.8 ± 1.8 min 35ºC = 29.6 ± 1.9 min	8
VanHaitsma et al. (194)	Crossover	20 Trained male cyclists (54.8 ± 5.9), Level 3	40 km cycling TT	TT in temperate (21ºC, 20% RH; CON) and heat (35ºC, 25% RH; INTER)	Time point unknown, including temperature in warm-up	21ºC = 75.2 ± 6.6 min 35ºC = 79.0 ± 7.2 min	8

Table 3.1 Continued

Study, year	Study Design	No. of participants and training status[a],	Exercise protocol	Intervention	Time point of environmental exposure or	Performance measurement (total time or mean power output (MPO) in watts)	PEDro Score

		Participant Classification Score			intervention manipulation		
PRE-COOLING STRATEGIES							
Barwood et al. (20)	Crossover	11 Trained male cyclists, Level 3	40 km cycling TT	TT in heat (32ºC, 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 ~22ºC), 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. (28)	Crossover	7 Recreational male cyclists, Level 2	30 min cycling TT	TT in the heat (32ºC, 60% RH) ingesting 900mL of hot (37ºC; CON) or cold fluid (2ºC; INTER) 10 min before trial	After fluid ingestion, 5 min at rest in heat, temperature of warm-up unknown	37ºC fluid = 261 ± 22 MPO 2ºC fluid = 275 ± 27 MPO	8
Duffield et al. (49)	Crossover	8 Moderate to well-trained male cyclists (lactate threshold 221 ± 42 W), Level 2	40 min cycling TT	TT in the heat (33ºC, 50% RH) with (INTER) and without (CON) lower body cold water immersion (CWI) at 14ºC 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 14ºC CWI = 198 ± 25 MPO	8
Gonzales et al. (71)	Crossover	10 trained male cyclists (59.1 ± 7.0 ml $kg^{-1}min^{-1}$), Level 3	20 min cycling TT	TT in the heat (30ºC, 79% RH) with (INTER) and without (CON) a cooling vest worn and refrigerated headband in warm-up in heat	18 min warm-up with cooling. 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. (6)	Crossover	15 Competitive male cyclists (397 ± 58 PPO), Level 3	20 km cycling TT	TT with correct fb (CON), positive deception (Pos-fb; INTER)	Informed travelled 25-m further than they had every km	CON = 28.4 ± 1.6 min Pos-fb = 28.6 ± 1.5 min	8

Table 3.1 Continued

Study, year	Study Design	No. of participants and training status[a], Participant Classification Score	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. (31)	Crossover	7 Recreational male cyclists (58.8 ± 5.7 ml $kg^{-1}min^{-1}$), Level 2	30 min cycling TT	TT in the heat without deception (31ºC, 64% RH; CON) and with (told the temperature was 26ºC, 60% RH, reality: 32ºC, 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. (91)	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 $kg^{-1}min^{-1}$), Level 3	16.1 km cycling TT	TT with accurate ride-alone fb (CON), against a pacer representing speed of CON (Neutr-fb; INTER), speed of pacer 102% above baseline, unware (Neg-fb; INTER) and a subsequent ride-alone trial post pacer (not included in meta-analysis)	From the onset of the trial	*Control Group:* CON = 27.1 ± 2.1 min Neutr-fb = 26.5 ± 1.6 min Inform-fb = 26.6 ± 1.6 min *Deception Group:* CON = 27.0 ± 1.3 min Pos-fb = 26.4 ± 1.1 min Inform-fb = 26.6 ± 1.4 min	9
Jones et al. (89)	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 $kg^{-1}min^{-1}$), Level 3	16.1 km cycling TT	TT with accurate ride-alone fb (CON), against a pacer representing MPO of CON (Neutr-fb; INTER), MPO of pacer 102% above CON, unaware (Neg-fb; INTER) and a subsequent ride-alone trial (Inform-fb; INTER)	From the onset of the trial	*Control Group:* CON = 26.3 ± 1.4 min Neutr-fb = 26.2 ± 1.3 min Inform-fb = 26.4 ± 1.3 min *Deception Group:* CON = 26.4 ± 0.5 min Pos-fb = 26.2 ± 0.4 min Inform-fb = 26.3 ± 0.5min	9
Shei et al. (158)	Crossover	14 Competitive male cyclists (61.6 ± 0.6 ml $kg^{-1}min^{-1}$), Level 3	4 km cycling TT	TT with accurate fb, ride-alone (CON), MPO of pacer 102% above baseline, unware (Neg-fb; INTER) and a subsequent ride-alone trial post deception (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

Table 3.1 Continued

Study, year	Study Design	No. of participants and training status[a], Participant Classification Score	Exercise protocol	Intervention	Time point of environmental exposure or intervention manipulation	Performance measurement (total time or mean power output (MPO) in watts)	PEDro Score
Smits et al. (162)	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 $kg^{-1}min^{-1}$), Level 3	20 km cycling TT	TT 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. (173)	Crossover	12 Competitive cyclists (56.6 ± 6.6 ml $kg^{-1}min^{-1}$), Level 3	40 km cycling TT	TT 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
Waldron et al. (197)	Crossover	9 Well-trained male cyclists (60.5 ± 3.3 ml $kg^{-1}min^{-1}$), Level 4	4 km cycling TT	TT with accurate 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 = 5.9 ± 0.7 sec Pos-fb = 6.2 ± 0.6 sec	9
William et al. (202)	Parallel group	22 untrained males in cycling, CON (n = 11) and No-fb (n = 11) groups (50 ± 9 ml $kg^{-1}min^{-1}$), Level 1	4 km cycling TT	TT with distance knowledge and 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; **Inform-fb** = informed of the nature of a previous trial; **INTER** = intervention trial; **fb** = feedback; **Pos-fb** = positive deceptive feedback; **Neg-fb** = negative deceptive feedback; **Neutr-fb** = neutral feedback; **No-fb** = no; **TT** = time-trial

3.4.2 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 (10, 34, 142). Mean power output was significantly reduced for all intervention trials (MD = -49.33 watts (W), 95% CI = -76.89 to -21.81, p = 0.000) compared to the control (normoxia) trial (*p* = 0.000, **Figure 3.3**). There was a non-significant trend for a small reduction in MPO for the start section (MD = -42.65 W, 95% CI = -109.31 to 24.01, p = 0.210) in the hypoxia trials. However, MPO was significantly reduced in 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 (FiO_2: 0.19), moderate (FiO_2: 0.16) and high (FiO_2: 0.14) stimulated altitude compared to sea level (34). 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 3.3**). One study (10) demonstrated a marked reduction in the Pacing Index Change Score (**Figure 3.12**) across the trial for the hypoxic trial IP (FiO_2: 0.15) relative to the control (normoxic) trial condition.

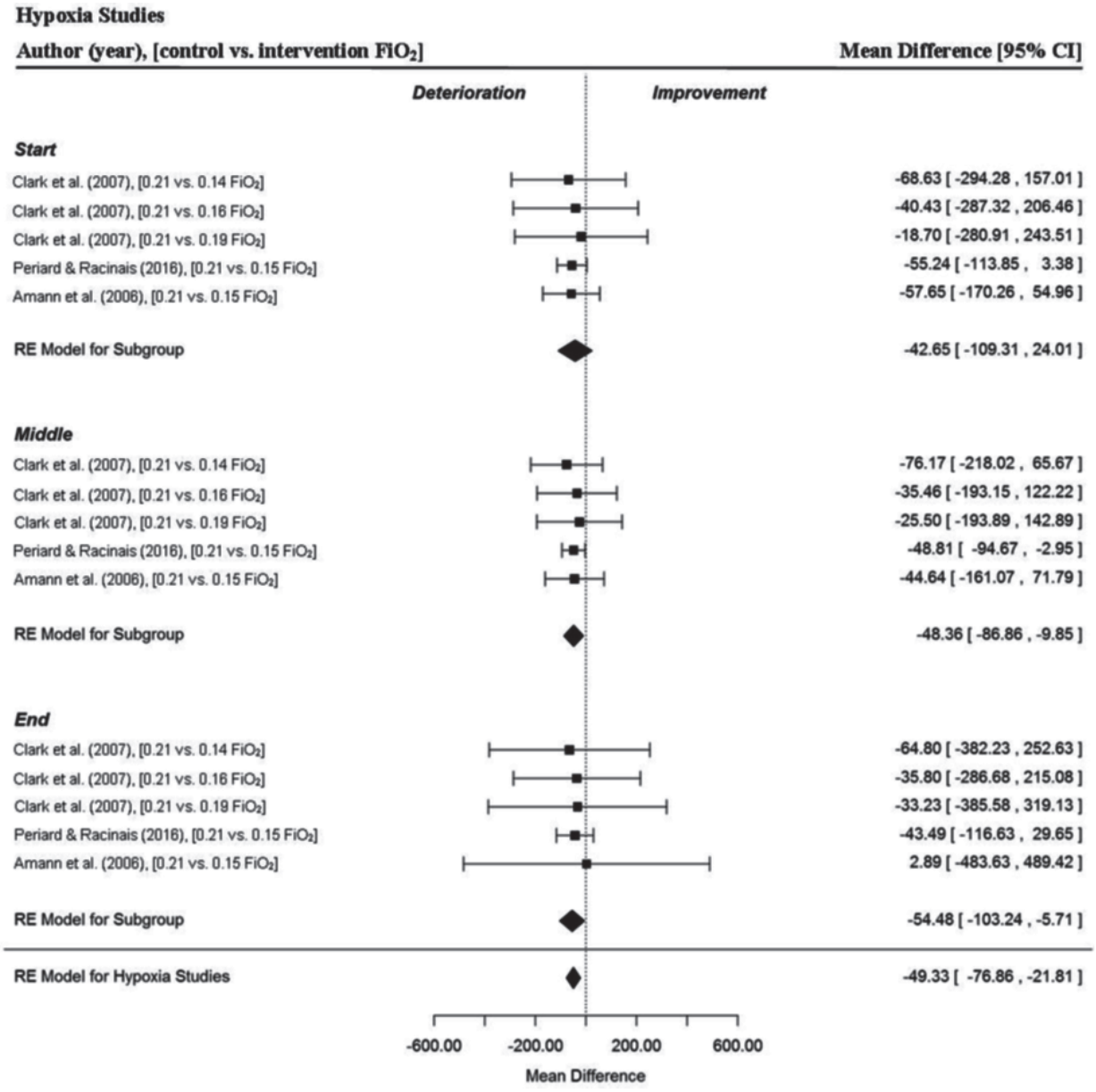


Figure 3.3 Forest plot for hypoxia meta-analysis illustrating power output during start, middle and end sections compared to normoxia trials. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

3.4.3 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 (10, 187). 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 3.4**).

3.4.4 Analysis of studies which investigated the manipulation of heat-stress

Heat-stress (**Figure 3.5**) 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 11 included studies (MD = -24.79 W, 95% CI = -39.52 to -10.06, p = 0.001) (5, 7, 31, 134, 139, 141, 142, 155, 175, 189, 194). 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 3.12). 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).

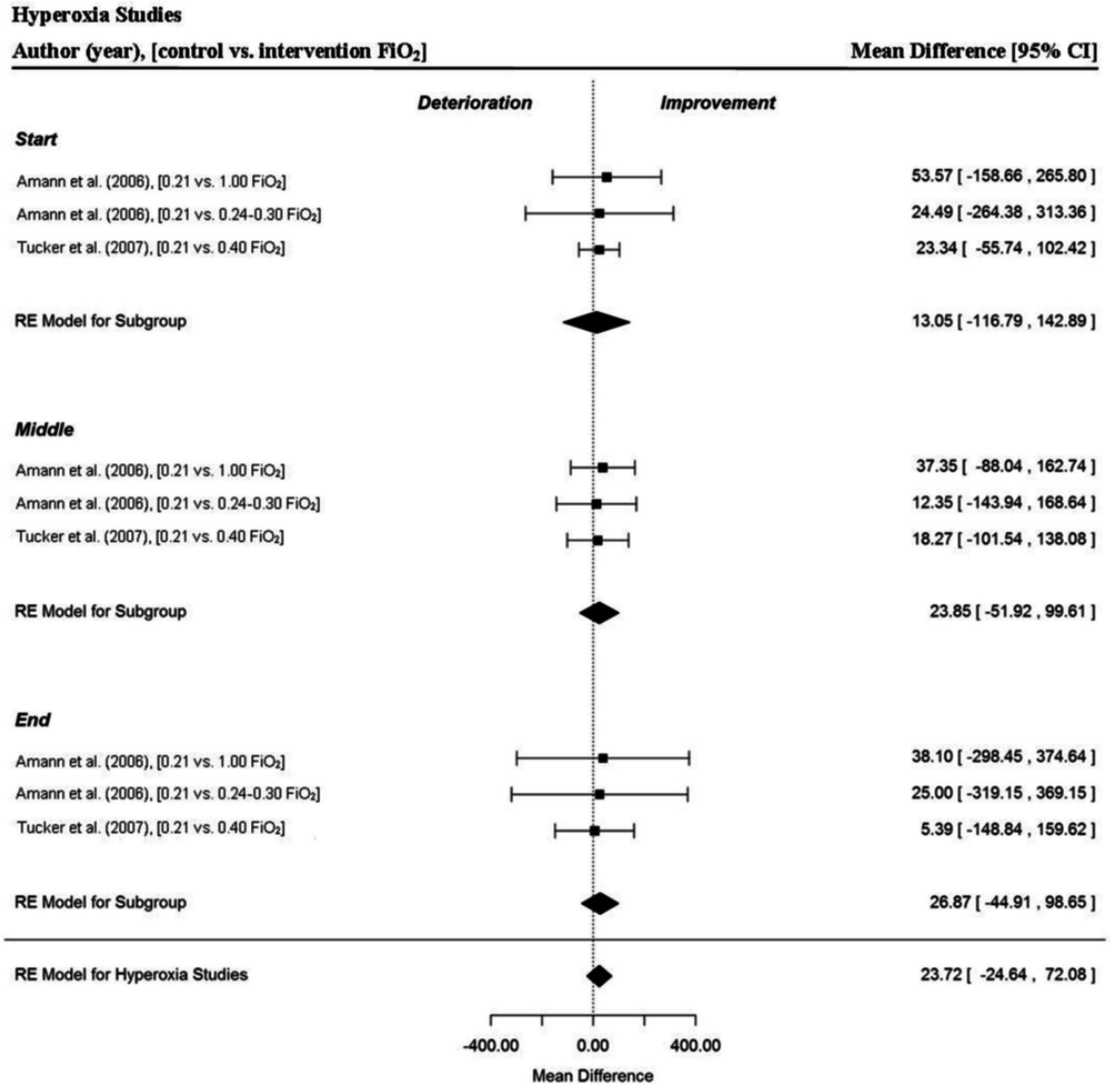


Figure 3.4 Forest plot for hyperoxia meta-analysis illustrating power output during start, middle and end sections compared to normoxia trials. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportion to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

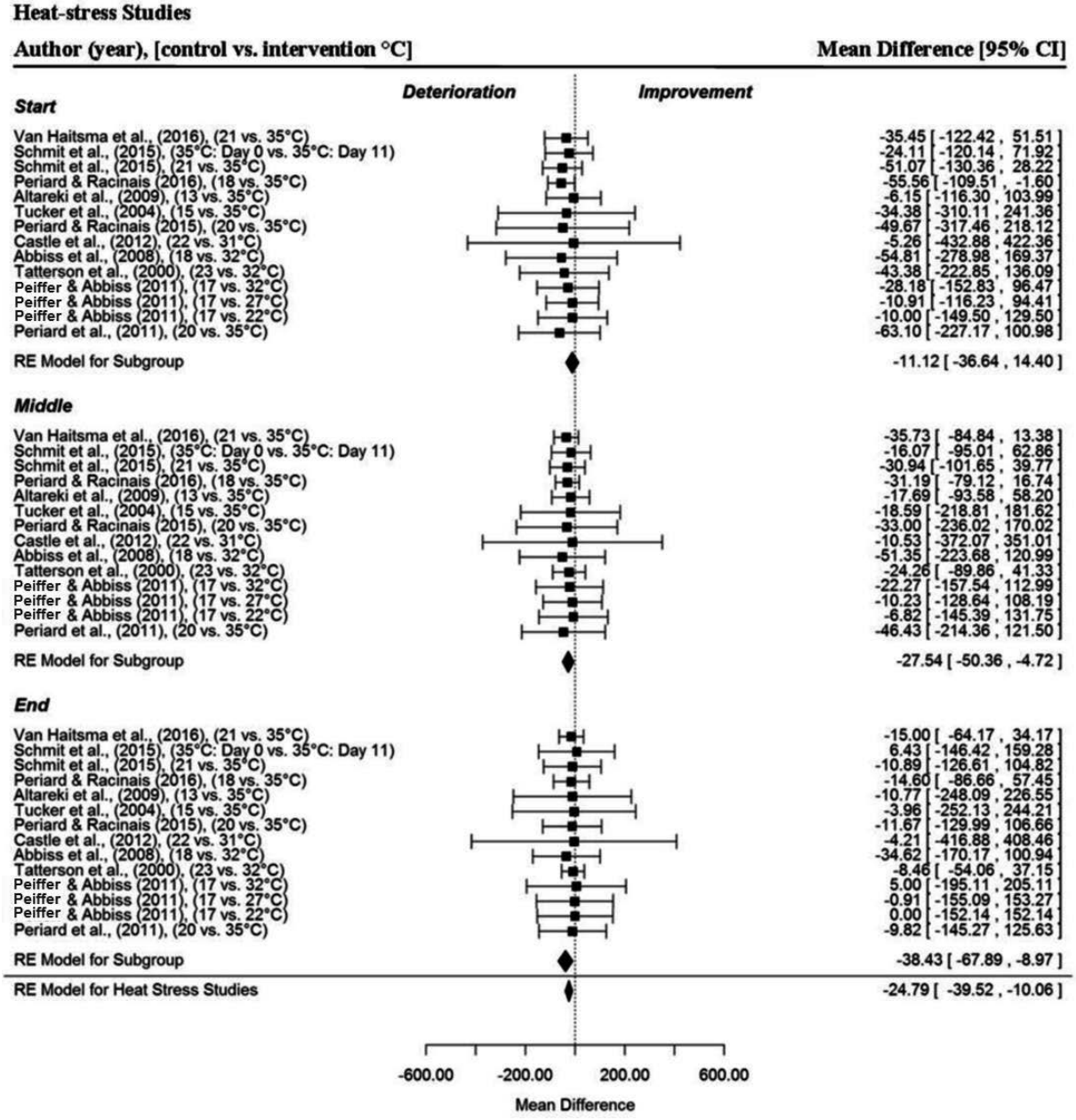


Figure 3.5 Forest plot for heat-stress meta-analysis illustrating power output during start, middle and end sections compared to temperate trials. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

3.4.5 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 (20, 28, 49, 71), 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 3.6**).

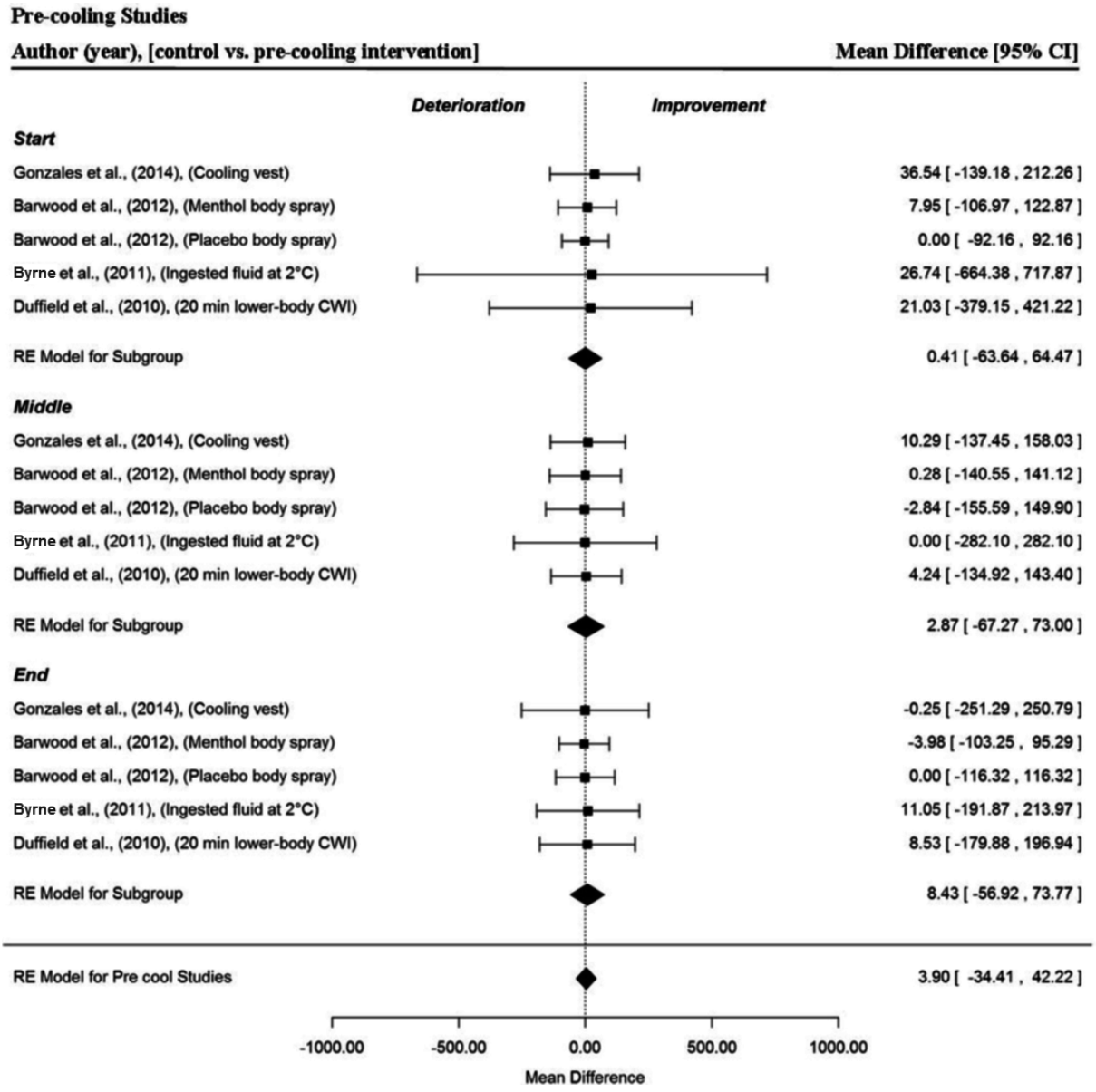


Figure 3.6 Forest plot for pre-cooling intervention meta-analysis illustrating power output during start, middle and end sections power output for start, middle and end sections compared to no intervention. Squares represent individual study mean difference and the lines represent 95% CIs. The size of 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.

3.4.6 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 (6, 31, 197). 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 3.7**). This was particularly evident, when cyclists were informed that their rectal body and environmental temperature were lower (0.3°C and 26°C, respectively) compared to control (31).

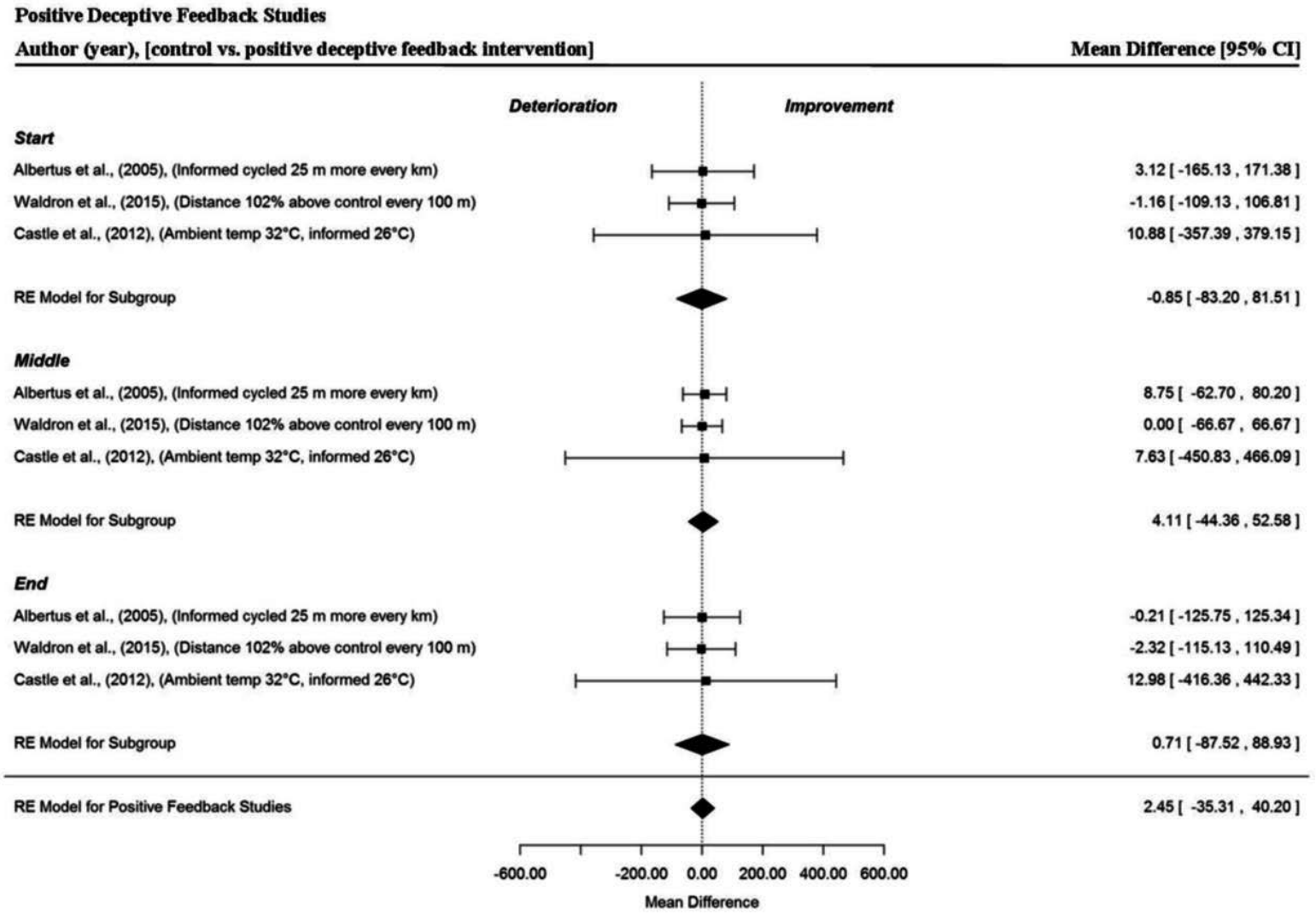


Figure 3.7 Forest plot for positive deceptive feedback meta-analysis illustrating power output during start, middle and end sections power output for start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size 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.

3.4.7 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 (89, 91, 158). 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 in overall performance and individual section MPO when participants were provided with negative deceptive feedback (**Figure 3.8**).

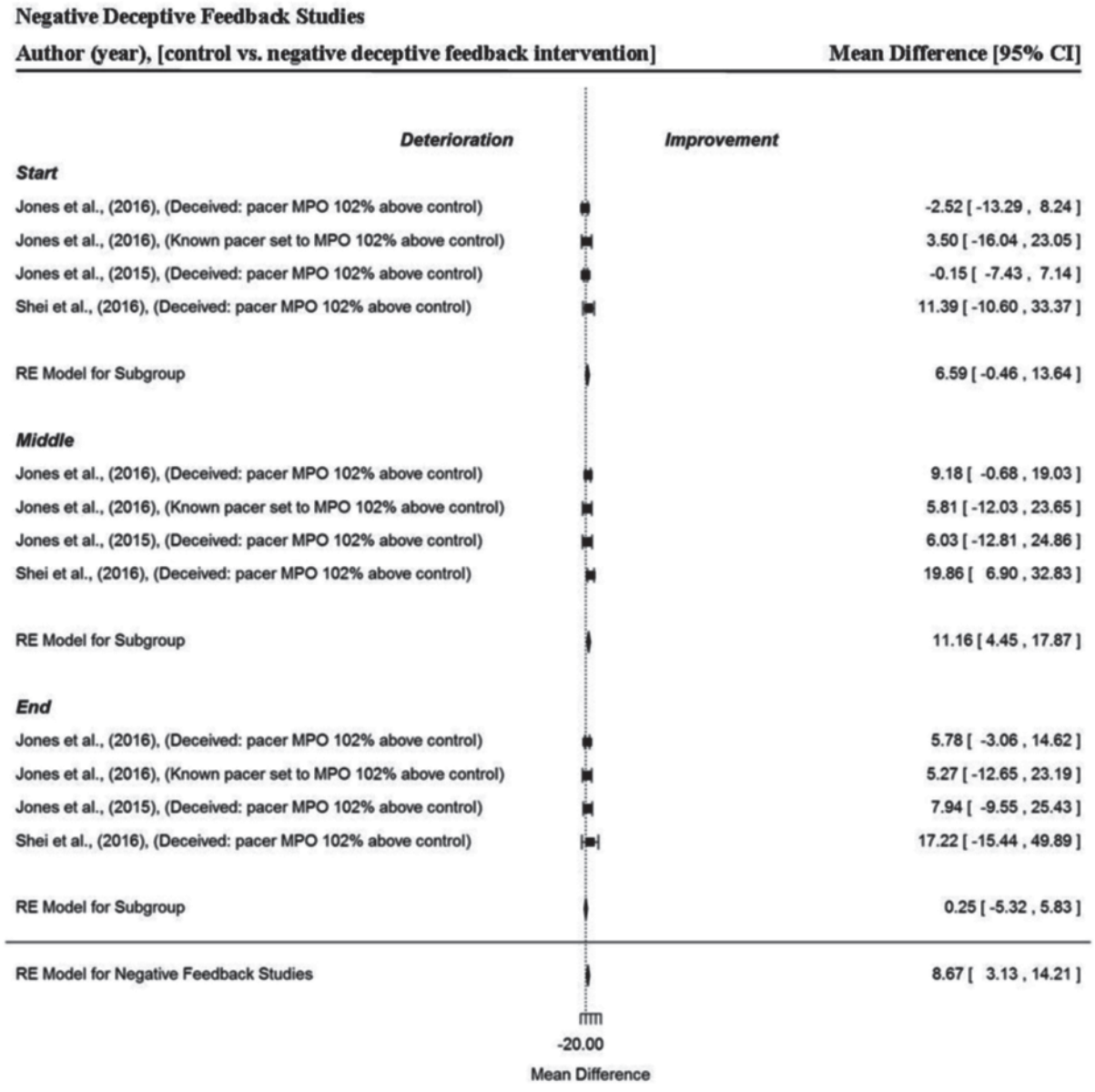


Figure 3.8 Forest plot for negative deceptive feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the 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.

3.4.8 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 (91, 197). **Figure 3.9** 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) or end (MD = -0.17 W, 95% CI = -14.39 to 14.05, p = 0.981) sections.

3.4.9 Analysis of studies which investigated the manipulation of no feedback

Data points were extracted from three different comparisons and three separate investigations (162, 173, 202). 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, **Figure 3.10**). 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 = 20.94 W, 95% CI = -26.05 to 67.93, p = 0.382) sections of trials.

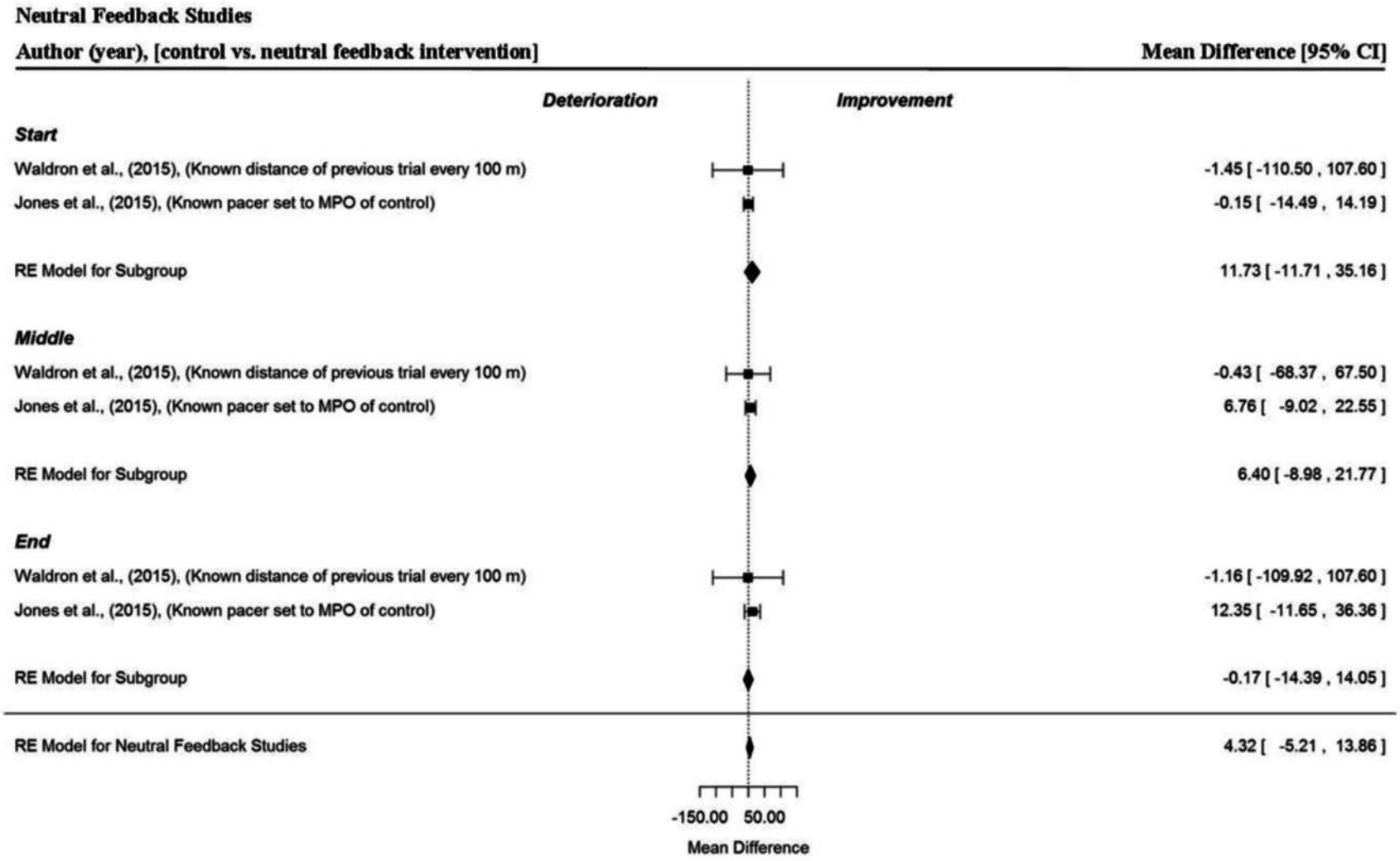


Figure 3.9 Forest plot for neutral feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

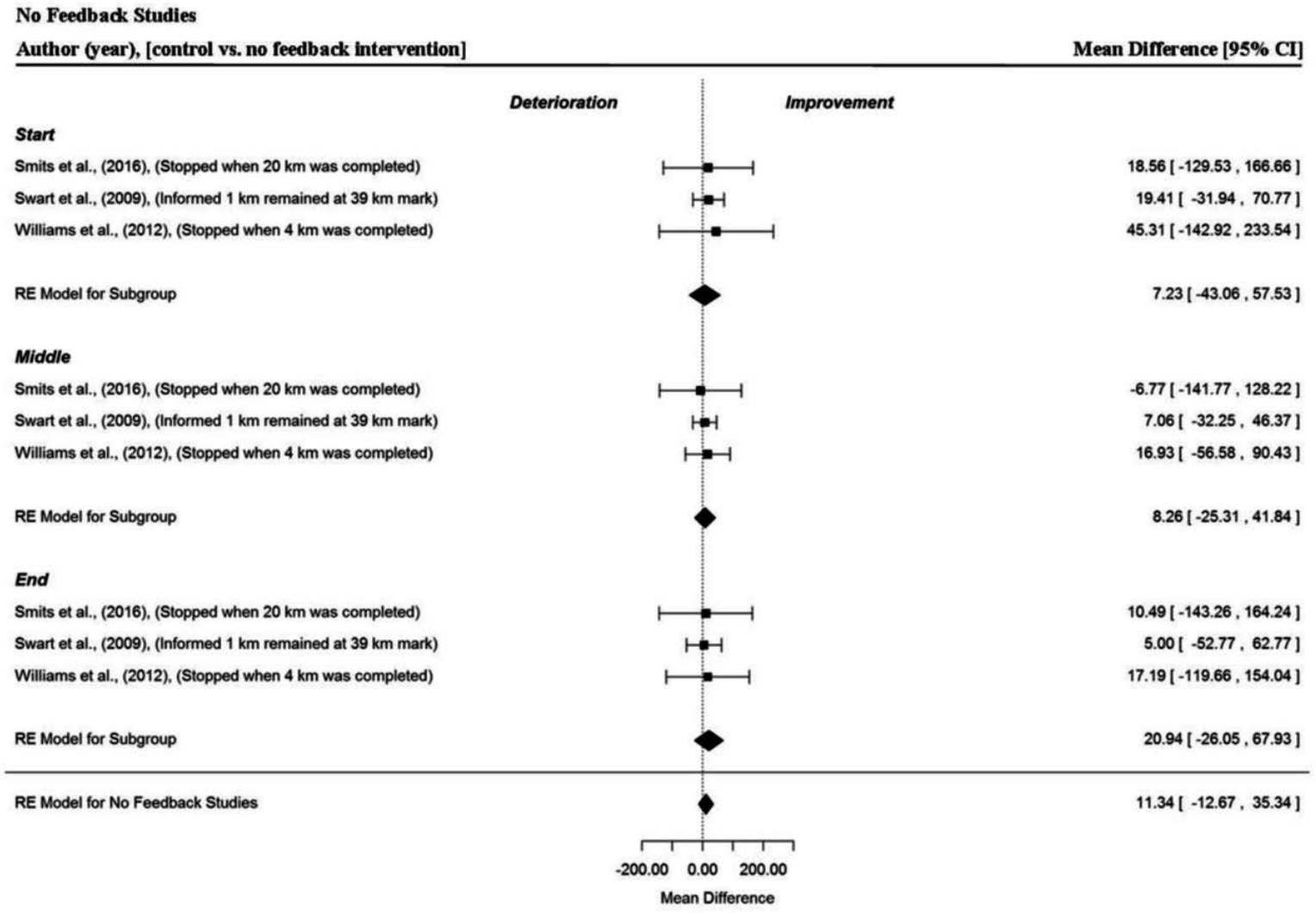


Figure 3.10 Forest plot for no feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

3.4.10 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 (89, 91, 158). 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, **Figure 3.11**). In these studies, participants were either unaware (89, 91, 158) or aware (89) of the increase MPO of their previous trial (102%), represented by a virtual on-screen avatar. Participants completed a subsequent (informed) trial with the presence of a pacer and known MPO increase (102%) (158) or with no pacer (89, 91). 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.

3.4.11 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 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, with the exception of the heat-stress group, the meta-analysis included a small sample in the defined themes, therefore the Q-test is likely underpowered for detecting true heterogeneity. Individual group results are outlined in **Table 3.1**

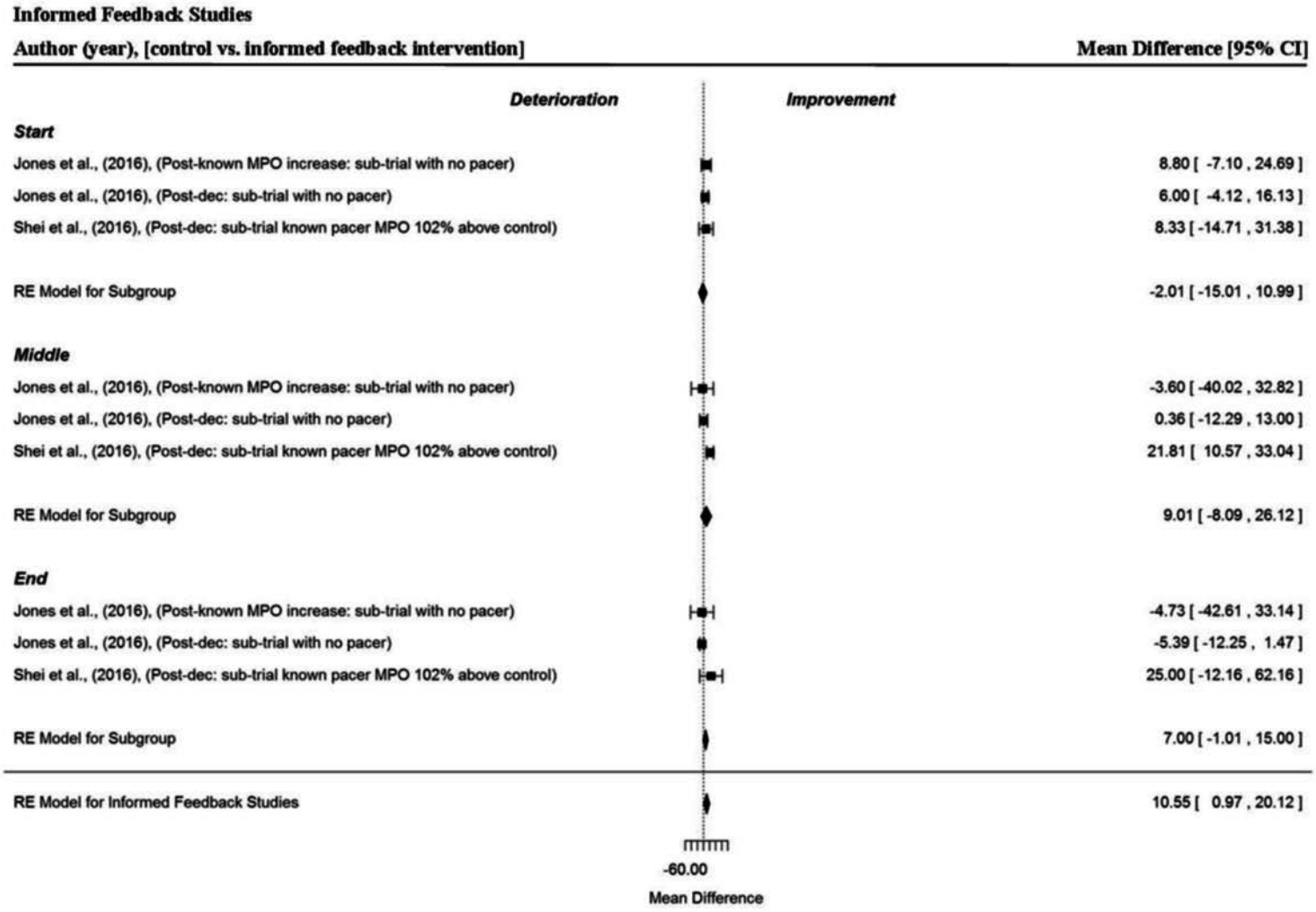


Figure 3.11 Forest plot for informed feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

3.5 DISCUSSION

Previously a number of reviews have been published concerning deception (90, 204), decision making (52, 118, 146, 161) and neurophysiological determinates (147) on 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.

3.5.1 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 ± SD: 16 ± 21 minutes) but were blinded 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 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 (78, 87). 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 FiO_2 disturbance, takes a significant amount of time to be assimilated and acted upon (78, 87, 108). A time lag spanning between 30 to 60 seconds from the start of the exercise bout before a pacing adjustment is made has previously been reported (10, 78, 87, 137). 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 (137), which may indicate 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 seconds 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 (87). 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 (137). 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 utilisation of the anaerobic capacity in the hypoxia trials, when integrated

within brain centres, might have led to the motor cortex reducing central motor drive to the exercising skeletal musculature 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 allocated time to deliberative decision-making. It has been hypothesised that incoming sensory afferent feedback, knowledge of the trial distance or duration (173), current momentary perceived effort influenced by prior experience (117), current expectations (such as outcome and strategic goals) (19, 146) and knowledge of current physical capacities (146) are integrated into decision making processes during self-paced exercise. At what point information resides in different brain centres and moves across sub-conscious, pre-conscious and conscious states is difficult to discriminate; however, if the participant consciously 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 (42, 120). 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 (111).

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 (34). In this study when well-trained cyclists' first (5-minute) time-trial was either at 3200-m or 2200-m simulated altitude, they demonstrated a conservative approach in the start section of

subsquent trials at lower simulated altitudes (200-m and 1200-m respectively). The authors attributed this observation to an order effect, and suggested 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. (10) study looks markedly different compared to the other studies (**Figure 3.12a**). 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.

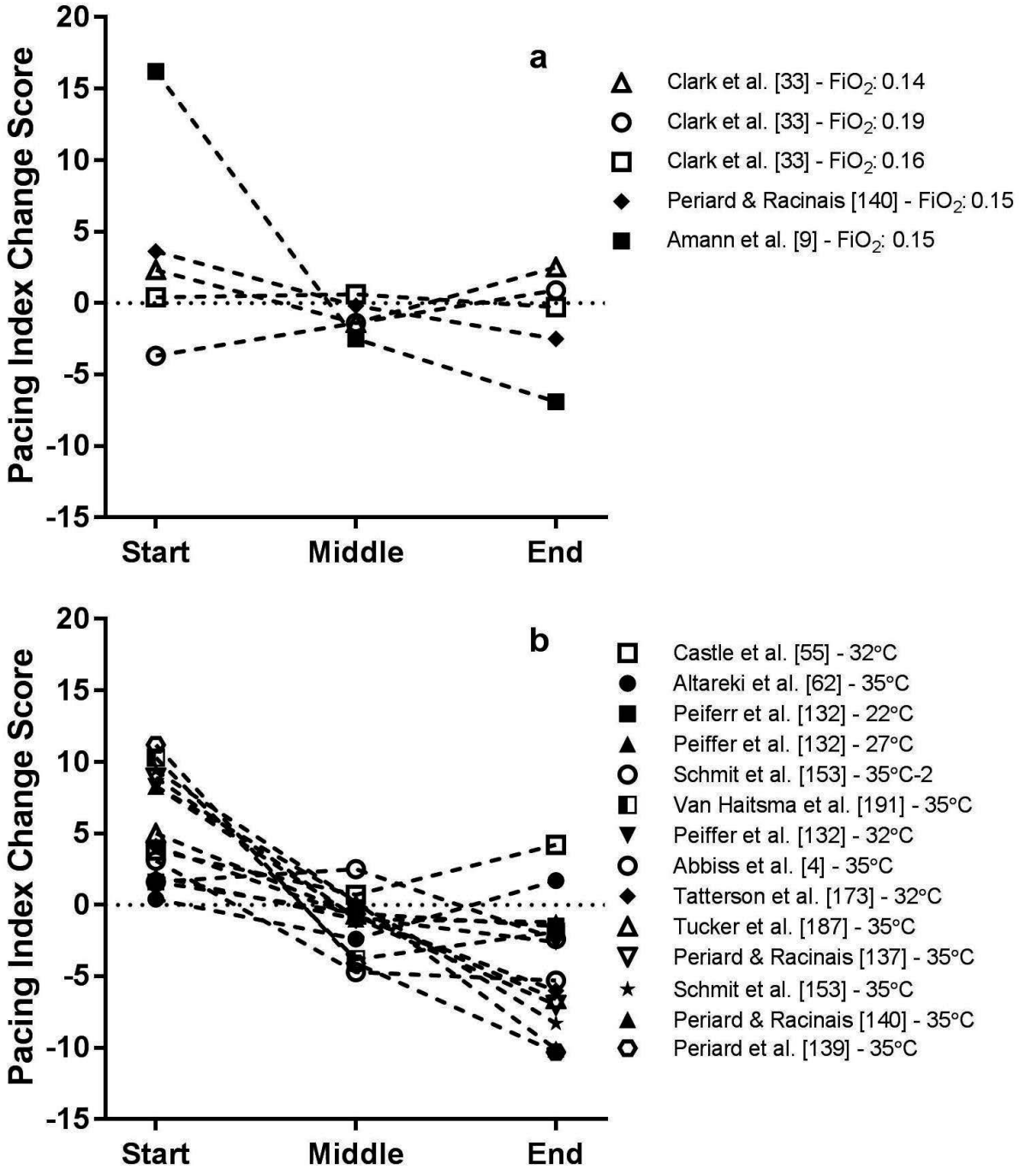


Figure 3.12 Exploratory graphical analysis of the pacing index change score difference between control and hypoxic condition (**a**) and control and heat-stress condition (**b**) for each individual segment. For an explanation of how the Pacing Index Change Score was calculated please refer to section 3.3.5 Data Analysis.

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 (10, 126, 135-138, 187). 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 (10, 187). Therefore, whether knowledge of breathing oxygen enriched air encourages a conscious decision to upregulate exercise intensity or not, compared to a normoxic trial, requires further exploration.

3.5.2 Pacing and heat-stress

When participants were exposed to hot and humid conditions the meta-analysis clearly demonstrated participants 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 minutes of exercise, despite

elevated skin temperatures, thermal sensation and perceived effort (53, 141, 175, 189). However, if participants are inexperienced at time-trialling in heat they can misjudge the start section of the trial by beginning the trial too aggressively (31, 154). Notably, for a number of the heat-stress trials, the Pacing Index Change Score exhibited was relatively greater and lower for the start and end sections, respectively, compared to the temperate condition trial (**Figure 3.12b**). This indicates an inability for participants to adopt an optimal pacing strategy from the onset of the trial in the hot condition, rather the pacing strategy has to be substantially adjusted as the trial progresses and 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 standardised warm-up) was ≤ 15 minutes in duration (31, 155, 194) or longer (5, 7). However, in the remaining studies either no mention was made as to participants being exposed to the heat-stress prior to the time-trials, or they did not expose their participants to heat-stress in the warm-up period (134, 139, 141, 142, 175, 189). 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 (155). 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-behavioural responses (153). 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 the body temperature data from these studies revealed that during time-trials lasting ≤ 30 minutes or where participants weren't exposed to heat up to 20 minutes before the trial, or both, there was little or no change evident in participants body temperature. This would suggest the adjustments observed in the pacing strategy were not necessarily caused by a change in body temperature but rather the changes in the pacing strategy alleviated significant changes in the body temperature. The potential cause 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, RPE and thermal sensation (155).

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 (80). This consequence appears to become more problematic in continuous exercise lasting over 30 minutes in duration, as the maintenance of blood pressure takes priority over blood flow to the skin and working musculature (30). 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 (86). This is due to the excessive heat-stress over the course of the trial, significantly impacting circulatory responses

which leads to a decrease in $\dot{V}O_2$ and subsequently a reduction in exercise intensity (142) and MPO as observed in this meta-analysis.

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. Firstly, whether the temperature of the control trial was in accordance with laboratory recommendations for a thermoneutral environment (18 to 23°C, relative humidity < 70%) (7, 134, 189). Secondly, 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. (7) was 22°C compared to 15°C by Periard and colleagues (141). It is possible that the relative temperature increase from cool (≤ 18°C) to hot (≥ 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 (7, 134); 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) (50). 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 ± SD: 19.2 ± 8.9 km h^{-1}). In addition, three studies did not mention whether convective cooling was used in heat trials (31, 155, 194). Differences in available air flow to dissipate heat may partly explain the variation of power output between individual studies.

3.5.3 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' (102, 125). 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 minutes of cold water (14ºC) or lower body immersion (49), ingesting 900 mL of cold fluid (2ºC) (28) or after wearing a cooling vest (18 minutes during warm-up) (71). 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 (30) during the early stages of exercise (~ 15 minutes) following pre-cooling strategies, although once exercise exceeds ~ 10 minutes, any physiological and perceptual changes induced by pre-cooling appears to dissipate (122). 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 minutes (M ± SD: 40 ± 22 minutes), and so the start section took a number of minutes to complete. However, no significant change in MPO for the start section of the trials was detected. A closer inspection of core temperature data from the original manuscripts revealed, with the exception of ingesting 900 mL of cold fluid (28), 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 (20, 49, 71). 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 minutes).

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 minutes) events (102). 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 (28, 49) 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 (32, 50). This was demonstrated by Dugas and colleagues (50) who restricted the fluid consumption of participants below *ab 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.

3.5.4 Pacing and feedback manipulations

Overall, positive, neutral and no feedback had no significant effect on overall or individual segment MPO. 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 (89, 91, 158). 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. (167) 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. (167) study. The meta-analysis also demonstrated *informed deception* (**Figure 3.11**; where participants were informed they had been deceived in a previous trial before completing a final trial with a known MPO increase) led to improved trial performance compared to an original control (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 improve 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 (157, 167). That said, 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.

3.5.5 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 (123, 124, 178). 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. 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 (139), only male participants were recruited in the included studies. Therefore the findings have an obvious sex bias, as unfortunately, there is a dearth of studies to date investigating changes in the pacing strategies exhibited by females.

3.5.6 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 (109) 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 sex, age or in the instance of a meta-analysis, between different studies of similar themes (101, 208).

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 or 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 judgement occurring from the start of the race. For athletes who compete globally, their race preparation needs to include practising 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 practise 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 *ab 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 or 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.

3.6 CONCLUSION

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.

4.0 STUDY TWO: HYPEROXIA AND INSPIRED GAS CONTENT DECEPTION HAS THE POTENTIAL TO CHANGE PACING AND PERFORMANCE DURING 4000-m CYCLING TIME-TRIALS.

Citation: **Davies, M. J**., Clark, B., Garvican-Lewis, L. A., Welvaert, M., Gore, C. J., & Thompson, K. G. (2018). Hyperoxia and inspired gas content deception has the potential to change pacing and performance during 4000-m cycling time-trials. *International Journal of Sport Physiology and Performance, Under Review.*

4.i FORM E: DECLARATION OF CO-AUTHORED PUBLICATION CHAPTER

For use in theses which include publications. This declaration must be completed for each co-authored publication and to be placed at the start of the thesis chapter in which the publication appears.

Declaration for Thesis Chapter 4

Declaration by candidate

In the case of Chapter 3 the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Study design, data collection and analysis and study write up	65

The following co-authors contributed to the work.

Name	**Nature of contribution**	**Extent of contribution (%)**	**Contributor is also a student at UC Y/N**
Bradley Clark	Assistance with study design, data collection, analysis and interpretation of results and proof reading of drafts	10	N
Laura Garvican-Lewis	Assistance with study design, interpretation of results and proof reading of drafts	5	N
Marijke Welvaert	Assistance with study design, analysis and interpretation of results and proof reading of drafts	5	N
Chris Gore	Assistance with study design, interpretation of results and proof reading of drafts	5	N
Kevin Thompson	Assistance with study design, analysis and interpretation of results and proof reading of drafts	10	N

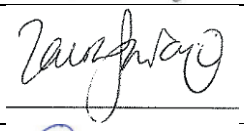
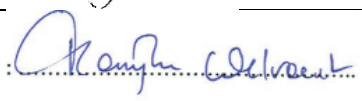
Candidate's Signature		**Date** 24/04/2018

Declaration by co-authors
The undersigned hereby certify that:

(7) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors.
(8) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
(9) they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
(10) there are no other authors of the publication according to these criteria;
(11) potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit; and
(12) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s)	Australian Institute of Sport Department of Physiology, Bruce, ACT Research Institute for Sport and Exercise, University of Canberra, Bruce ACT

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant.]

Co-author Signature Number	**Co-author's Signatures**	**Date**
Signature 1		24/04/2018
Signature 2		24/04/2018
Signature 3		24/04/2018
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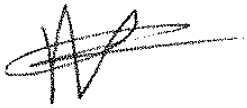
Signature 5		24/04/2018

4.1 ABSTRACT

Background: We sought to determine if a series of trials with fraction of inspired oxygen (FiO_2) content deception could improve 4000-m cycling time-trial (TT) performance.

Method: Fifteen trained male cyclists (mean ± SD: stature 181 ± 6.7 cm; body-mass 74.2 ± 8.0 kg; peak oxygen uptake 62 ± 6 $mL \cdot kg^{-1} \cdot min^{-1}$; maximal aerobic power 4.7 ± 0.4 $W \cdot kg^{-1}$) completed six, 4000-m cycling TT's in a semi-randomised order. After a familiarisation TT, cyclists were informed in two trials they were inspiring normoxia (NORM, FiO_2: 0.21), however in one trial they inspired hyperoxia (NORM-DEC, FiO_2: 0.36). Similarly, during two subsequent TT's, cyclists were informed they were inspiring hyperoxia (HYPER, FiO_2: 0.36), but inspired normoxic air in one trial (HYPER-DEC). In the final TT (NORM-INFORM) the deception was revealed and cyclists were instructed to reproduce their best TT while inspiring normoxia.

Results: Greater MPO's and faster performance times were recorded when cyclists inspired hyperoxia irrespective of whether participants were aware (HYPER) or not (NORM-DEC) compared to NORM ($P < .001$). However, performance was only found to improve for NORM-INFORM (377 W [95% CI 325, 429]) vs NORM (352 W [299, 404]), $P < 0.001$) when participants (n = 4) completed the trials in the following order: NORM-DEC, NORM, HYPER-DEC, HYPER.

Conclusions: Short-term cycling performance improved with acute exposure to hyperoxia. Mechanisms for the improvement were likely physiological, however improvement in a deception trial suggests an additional placebo effect may be present. Finally, a particular sequence of oxygen deception trials may have built psycho-physiological belief in cyclists such that performance improved in a subsequent normoxic trial.

Keywords: pacing, exercise reserve, hyperoxia, deception, feedback

4.2 INTRODUCTION

Pacing strategies are often defined as pre-determined 'templates' which are built through prior experience and recalled for future performances (117). Once exercise commences, incoming sensory information is evaluated to determine if the current work-rate should be maintained or adjusted, ensuring an acceptable pace is adopted (70). Recently, researchers have manipulated athlete expectations and beliefs regarding their abilities to examine how performance can be enhanced through access of reserve capacities (120, 204). To achieve this, deceptive information is provided, therein manipulating perceptual, environmental or performance feedback via non-invasive and practical methods (120). The purpose of such deception is to create uncertainty within the pacing template, causing athletes to deviate from their routine strategy (204). However, results from a recent meta-analysis suggest changes in pacing and performance is largely dependent on the type of deception received (40).

Studies have delivered deceptive information by informing athletes they are performing better (positive) or worse (negative) via inaccurate performance information (40). Negative deceptive feedback, specifically performance deception, appears the most effective form of deception to elicit performance improvements (40). In performance deception trials, athletes have raced against a virtual on-screen pacer whom they believed was programmed to mimic mean power output (MPO) from their previous best performance. In reality, the pacer was programmed with a MPO which was +2% greater (40, 91, 158, 169). Interestingly, performance improvements in a negative performance deception trial, may also be retained in a subsequent TT once deception is revealed (158, 169). These improvements are believed to be the result of athletes accessing their reserve capacities, likely the product of a greater anaerobic energy contribution in later stages of a trial (169). However, whether deception regarding environmental conditions can elicit similar responses is largely unknown.

Environmental deception is designed to change athlete task expectations such that they change their pacing approach. A challenge for researchers providing deceptive environmental information is whether the deception is subtle enough to be effective. Previous research has examined the effects of deceptive information pertaining to ambient temperature (31). However, deceiving athletes of the fraction of inspired oxygen (FiO_2) may be more successful, as it would likely be harder to detect than a change in ambient temperature (40). Previous studies blinding athletes to the FiO_2 indicate even slight manipulations are sufficient to influence cycling performance (78, 87). Specifically, self-paced cycling exercise completed in hyperoxia (FiO_2: > 0.21) has demonstrated to improve performance by 3–14% compared with normoxic trials (10, 105, 137, 187). However, to our knowledge, no current research describes the effects of FiO_2 deception on athletic performance. Therefore, whether knowledge of inspiring hyperoxia encourages an upregulation of exercise intensity compared to a normoxic trial requires further exploration. Moreover, whether a series of oxygen content deception trials can build psycho-physiological belief in cyclists to improve performance in a subsequent trial in normoxia once deception has been revealed (similar to racing against a manipulated, virtual, on-screen pacer), remains unknown.

The aim of the present study was to determine if a series of trials manipulating FiO_2 and belief (through deception) might change the pacing profile and performance during 4000-m cycling TT's. Collectively this information will further our understanding of how hyperoxia and deceptive information alters trained cyclists exercise regulation and further, determine if these techniques could be used in training to enhance performance.

4.3 METHODS

4.3.1 Study Design:

Cyclists completed six 4000-m cycling TT's at least 48 hours apart over two weeks (**Figure 4.1**). In trials one to three, cyclists were informed they were inspiring normoxic air (FiO_2: 0.21 ± 0.04): familiarisation (FAMIL), normoxic (NORM) and normoxic deception (NORM-DEC). However, in NORM-DEC cyclists were actually inspiring hyperoxic air (FiO_2: 0.36 ± 0.01). Cyclists then completed two informed hyperoxic trials (HYPER, FiO_2: 0.36 ± 0.01): hyperoxia (HYPER) and hyperoxia deception (HYPER-DEC). However, in HYPER-DEC cyclists were actually inspiring normoxic air as per the NORM trial. 'NORM' and 'HYPER' trials were completed in a double-blinded and randomised order, meaning cyclists completed trials in one of four possible orders: HNHN (i.e. NORM-DEC, NORM, HYPER, HYPER-DEC; n = 4), HNNH (n = 4), NHHN (n = 3) and NHNH (n = 4). A sixth and final trial was then completed, where all cyclists were informed they had been deceived, and were instructed to reproduce their best trial performance whilst inspiring normoxic air (NORM-INFORM, FiO_2: 0.21 ± 0.04). All participants received a one-page factsheet before participating and were briefed throughout the study on the ergogenic benefits of inspiring hyperoxia on performance [Appendix 2].

4.3.2 Participants:

Fifteen trained male cyclists (mean ± SD: stature 181 ± 6.7 cm; body-mass 74.2 ± 8.0 kg; peak oxygen uptake ($\dot{V}O_{2peak}$) 62 ± 6 $mL \cdot kg^{-1} \cdot min^{-1}$; maximal aerobic power (MAP) 4.7 ± 0.4 $W \cdot kg^{-1}$) volunteered to take part in the study. Cyclists were instructed to maintain their normal diet, refrain from strenuous exercise, caffeine or alcohol in the 24 hours preceding each test.

All cyclists provided informed consent before participating. The study was approved by the University of Canberra Research Ethics Committee.

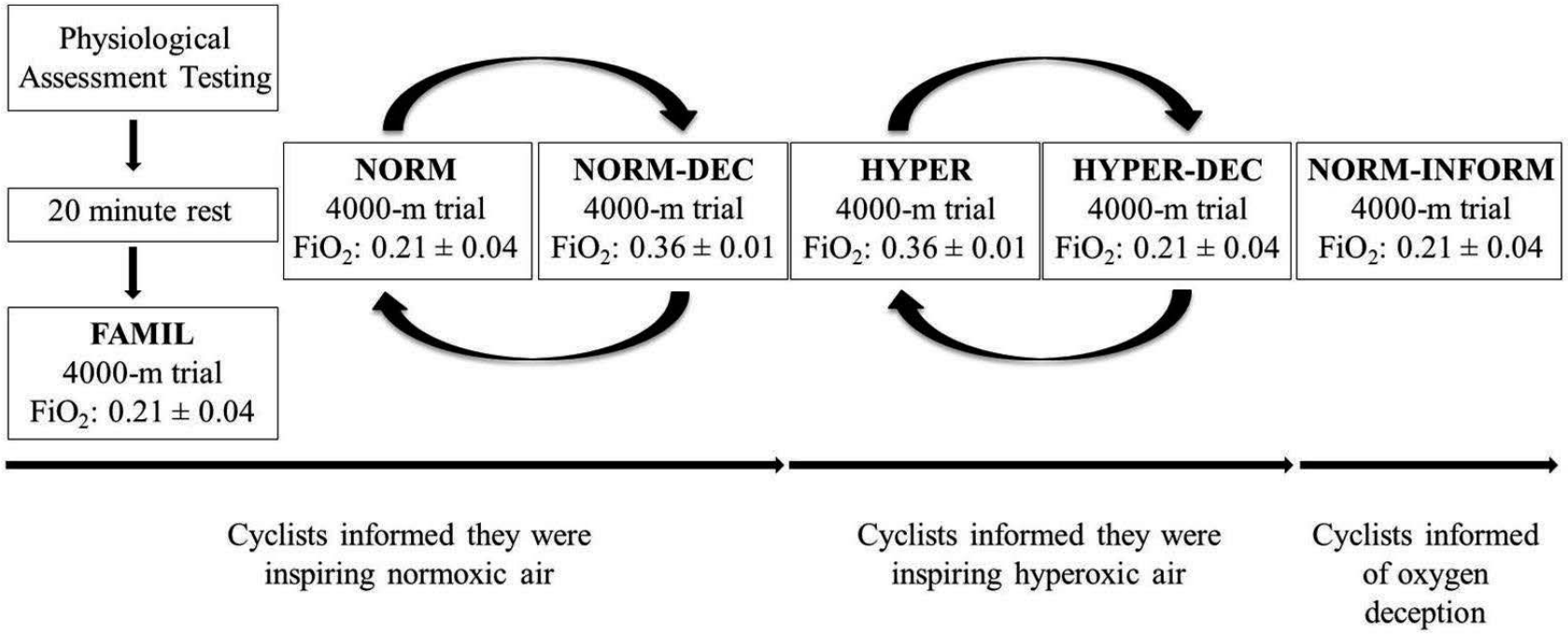


Figure 4.1 Study design including actual and informed FiO_2 for each trial. Randomised trials are indicated by black arrows. Both the cyclists and researcher (who conducted the experimental trials) were blinded to the actual FiO_2.

4.3.3 Experimental Procedures

Preliminary Testing:

All TT's were completed on a VelotronDynafit Pro cycle ergometer (RacerMateInc, CITY, WA, USA) with power output (PO) sampled at 1 Hz Prior to experimental trials, cyclists completed a graded exercise test (GXT) to volitional exertion for determination of $\dot{V}O_{2peak}$ and maximal aerobic power (MAP). After a 10-minute self-paced warm-up, PO began at 150 W and increased by 25 W every three minutes until volitional exhaustion or when cadence dropped below 80 revolutions per minute. Respiratory gases were collected using a custom-built, open-circuit, indirect calorimetry system with associated in-house software for determination of oxygen uptake (151). The highest $\dot{V}O_2$ averaged over a 30-second period was defined as $\dot{V}O_{2peak}$. MAP was determined as the final completed stage plus the proportion of any uncompleted stage attempted during the GXT (96). Heart rate (HR) was recorded continuously during trials using a portable telemetry device (Polar s810i, Kempele, Finland). Before each test, the ergometer was adjusted to replicate each cyclist's own racing bicycle, including crank length, handlebars, saddle and pedals. These measurements remained consistent for subsequent trials.

4000-m time-trial protocol:

Before each TT, cyclists were seated for 10 minutes to complete a 'wash in' phase where the relevant gas mixture of the specific trial was inspired. The purpose of the wash in phase was to acclimate cyclists to the administered hyperoxic gas before completing an 11-minute standardised warm-up. Briefly, cyclists completed four minutes at 60% MAP, three minutes at 70% MAP, two minutes at 80% MAP, one-minute building from 80-90% MAP, a six second sprint, finishing with one-minute of active recovery (113). Following the last stage of

the warm-up, cyclists had one-minute of passive recovery before commencing the TT. Before each trial the primary researcher instructed cyclists to give a maximal effort and complete the TT as fast as possible. Performance time, PO, cadence and HR were recorded at one second intervals, before assigning data to segments representing 10% of the distance completed for each trial. To describe the variability in the pacing strategy between trials we also calculated the pacing index using the following equation (208).

(*Segment mean power output*/ *Overall trial mean power output*)∗ 100

Constant visual feedback was provided regarding distance and gear selection via a large television screen paired with the cycling ergometer, providing a visual representation of cyclists completing a virtual flat 4000-m course. However, all other feedback including elapsed time, PO, velocity, cadence and HR was withheld. Cyclists were free to self-select cadence, gear ratio and vary cycling posture throughout. Cooling was provided by a fan placed one meter in front of cyclists (~20-km.h^{-1}). Finally, verbal encouragement was given by the primary researcher from a pre-prepared script at 500-m intervals and at 250-m and 100-m to completion (74).

Environmental manipulation:

During trials two to five, the primary researcher was blinded to the inspired FiO_2. To achieve this, another researcher filled two large bags (1200 L) with either hyperoxic air, which was produced by mixing normal room air with medical grade oxygen (O2 concentration 100%, BOC Gases and Gear, Canberra, Australia), or ambient air for normoxic trials. Both bags were filled simultaneously to the desired volume and gas content was sampled before each trial (normoxia actual: FiO2 0.21 ± 0.04; hyperoxia actual: 0.36 ± 0.01). Gas for all conditions was inspired through the bags, directed by a one-meter tube and delivered via a Hans Rudolph one-way valve (Hans Rudolph Inc, Shawnee KS, USA). The level of

hyperoxia was selected based on available literature where a subtle increase in the inspired oxygen content (0.35 – 0.40 FiO_2) can improve performance but be difficult for participants to detect when blinded (163). In addition, the administered hyperoxic air was based on recommendations from the Australian Institute of Sport (AIS) Medical Team on safe and ethical use of hyperoxic gas.

Electromyography and maximal voluntary contraction:

To assess muscle activity and fatigue across trials, surface electromyography (sEMG) was measured before, during and post-trials. To achieve this, five minutes before beginning the 'wash in' phase, cyclists performed three, five-second maximal voluntary contractions (MVC) as described previously (189). Briefly, after a three minute warm-up of easy pedalling, cyclists completed MVCs of the right leg extensors, on a 30-second cycle, at zero, 30 and 60 seconds. The same procedure was repeated one minute after each TT. Before each contraction cyclists were instructed to "push" against the ankle strap as hard as possible to provide an external focus, previously suggested to elicit greater total force production (74). Surface electromyography from cyclists right vastus lateralis leg muscle was recorded during all MVCs and continuously during each TT. The skin over the muscle belly was shaved, lightly abraded and cleaned, before the placement of two electrodes positioned on the midline belly of the muscle, inter-electrode distance of 30-mm (33). An electronic goniometer (ADInstruments, NSW, Australia) was positioned and secured on the lateral side of the right knee to set a 100° angle of knee flexion in the pre- and post- MVC, and secondly to trigger the corresponding sEMG at 100° knee flexion (99.7 ± 0.1°) during each downward pedal stroke during TT's. Raw sEMG and goniometer signals were sampled at 2,000-Hz via LabChart v8.1 (ADInstruments, NSW, Australia) system, filtered, rectified and smoothed to produce a linear envelope (or integrated EMG, iEMG), as described previously (33). Thus,

cycling sEMG data were amplitude-normalised, using the TT peak iEMG value at 100° knee flexion, normalised to the greatest peak iEMG value associated with the highest force produced in the pre-MVCs.

Psychological questionnaires:

Cyclists completed the motivational component of the Dundee Stress State Questionnaire, to measure success motivation and intrinsic motivation before each trial.(115) In addition, a Sports Emotion Questionnaire was completed after each trial as a sport-specific measure of post-competitive emotion to assess: anger, anxiety, dejection, excitement and happiness (92).

4.3.4 Statistical Analysis

Data were analysed with a General Linear Mixed Model which included fixed effects for trial order, and condition and segment. All models were estimated using Restricted Maximum Likelihood as implemented in the R package lme4 (21, 179). A random intercept for Subjects was included to account for intra-individual dependencies and inter-individual heterogeneity. Model assumptions were assessed through visual inspection of residual plots revealing no obvious deviations from homoscedasticity. However, heavier tails than would be expected were found under the normal distribution. To account for this, bootstrapped confidence intervals were obtained to minimize distributional impact. P-values were obtained using Type II Wald F tests with Kenward-Roger degrees of freedom as implemented in the R package car (62). Statistical significance was accepted at $P < .05$. Data are reported as mean estimates and 95% confidence intervals (CI).

4.4 RESULTS

4.4.1 Time-trial performance and pacing:

No Trial Order by Condition by Segment interaction was found for MPO ($F_{135,649}$ = 0.806, P = .939). However, Trial Order by Condition ($F_{15,649}$ = 3.216, P < .001, **Figure 4.2**) and Trial Order by Segment ($F_{27,649}$ = 4.680, P <.001, **Figure 4.3a**) interactions for MPO were significant. Similarly, a Trial Order by Condition interaction ($F_{15,649}$ = 2.616, P < .001) was found for performance time, however, Trial Order by Segment was not significant ($F_{27,649}$ = 0.637, P = .924). Specifically, cyclists who completed order: HNNH improved performance in NORM-INFORM (377 W [95% CI 303, 505]), and HYPER-DEC (369 W [296, 477]) compared to NORM (352 W [275, 436], **Figure 4.2d**). Additionally, across all groups, significantly greater MPO's and faster performance times were recorded when cyclists inspired hyperoxia compared to NORM, irrespective of whether they were aware (HYPER: 31 W, P < .001) or not (NORM-DEC: 25 W, P < .001, **Figure 4.2**). Finally, a significant Trial Order by Condition ($F_{15,649}$ = 6.276, P < .001, **Figure 4.4**) interaction was found for cadence, although Trial Order by Segment was not significant ($F_{27,649}$ = 0.104, P = .104).

No Trial Order by Condition by Segment interaction was found for the pacing index ($F_{135,649}$ = 0.955, P = 0.622). Similarly, Trial Order by Condition was not significant ($F_{15,649}$ = 0.000, P = 1.00, **Figure 4.2**). However, a significant Trial Order by Segment interaction was found ($F_{27,649}$ = 5.461, P < .001), where a faster starting strategy for all TT's in order HNHN and an end spurt initiated earlier in order HNNH were found compared to other groups (**Figure 4.3b**).

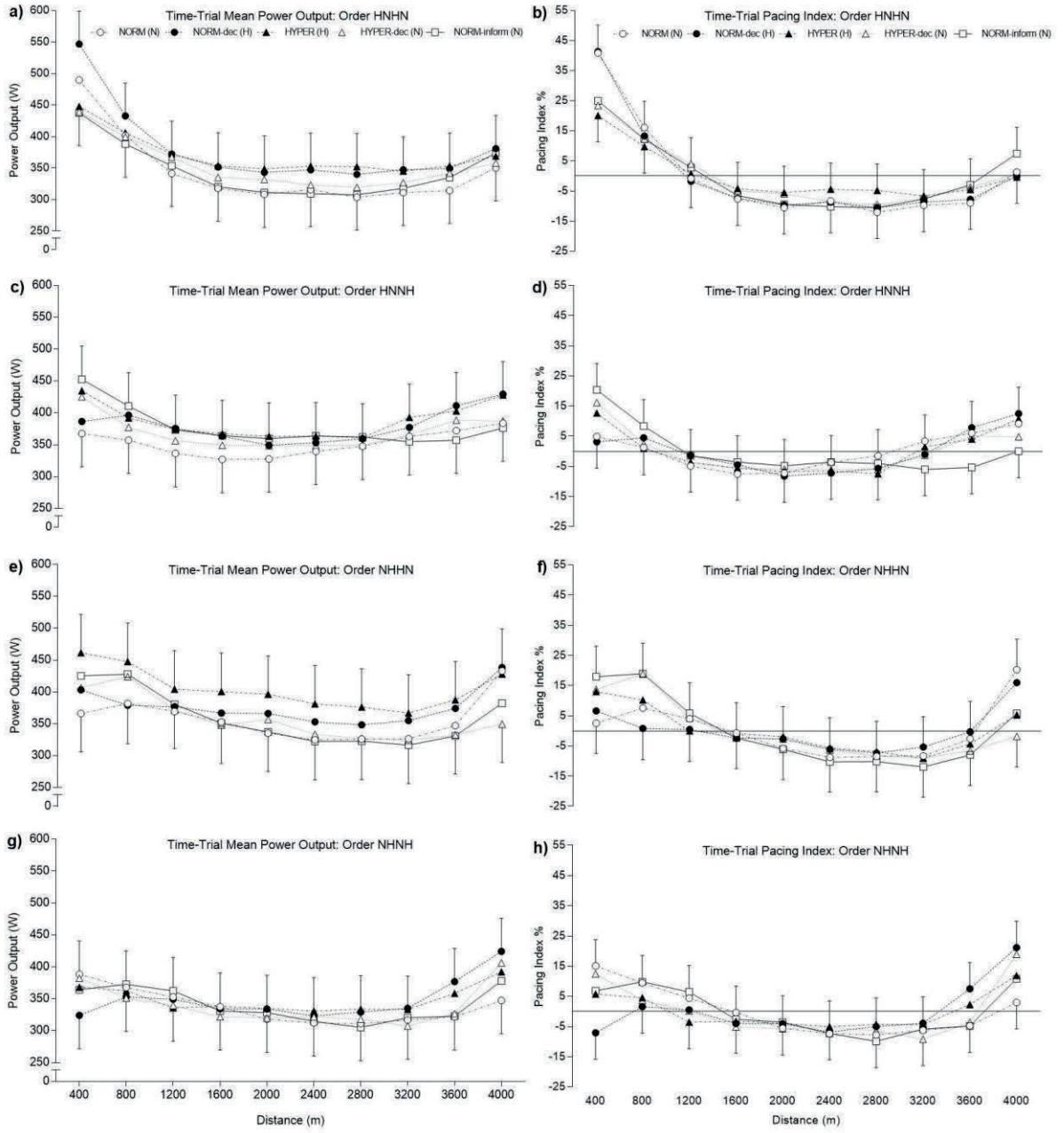


Figure 4.2 Mean power output (**left panel**) and associated pacing index (**right panel**) at 400-m intervals for each order group during 4000-m time-trials. Results are reported as mean estimates ± 95% confidence intervals.

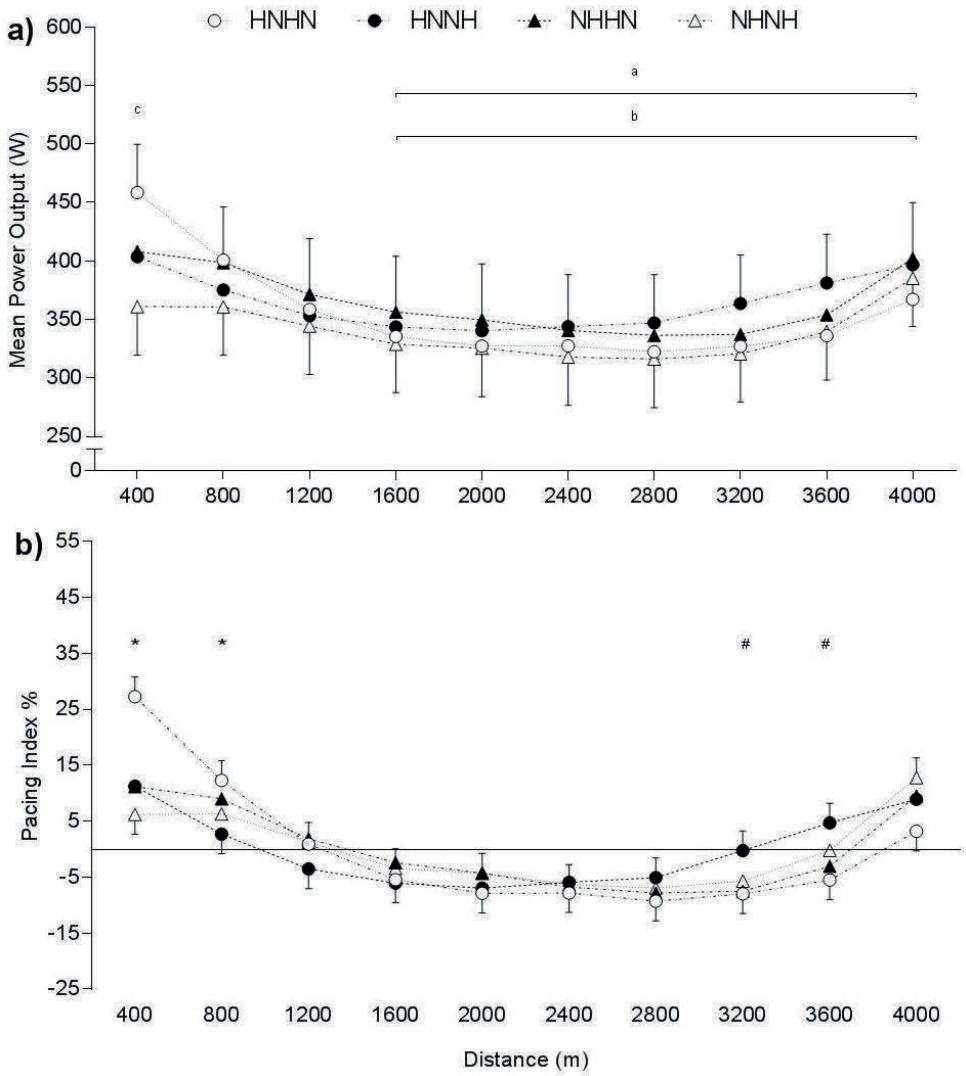


Figure 4.3 Mean power output for Trial Order by Segment interaction within the four trial orders (**a**) and associated pacing index (**b**) at 400-m intervals during 4000-m time-trials. [a] Significant difference between HNHN and HNNH. [b] Significant difference between NHHN and NHNH. [c] Significant difference between HNHN and NHHN. *Significant difference for HNHN compared to other orders. # Significant difference for HNNH compared to other orders. Results are reported as mean estimates ± 95% confidence intervals.

4.4.2 Heart rate:

Trial Order by Condition ($F_{15,609}$ = 9.241, P < .001, **Figure 4.4**) and Segment ($F_{27,609}$ = 3.658, P < .001) interactions were significant for HR. Heart rates were significantly higher during NORM-INFORM (186 bpm [157, 200]) compared to NORM (180 bpm [157, 197]) for order HNNH (**Figure 4.4c**). In contrast, the HR response was similar across all other conditions and orders.

4.4.3 Muscle activation during time-trials:

Trial Order by Condition interactions for iEMG ($F_{15,566}$ = 17.168, P < .001) and ratio of PO to iEMG were significant ($F_{15,566}$ = 20.105, P < .001). In contrast, no Trial Order by Segment for iEMG ($F_{27,566}$ = 0.285, P = 1.000) or ratio of PO to iEMG ($F_{27,566}$ = 0.124, P = 1.00) was found, where muscle activity followed a similar pattern across segments despite a decrease or increase in activation relative to FiO_2 (**Figure 4.5**).

4.4.4 Voluntary force production and muscle activation:

No significant Time by Trial Order by Condition interaction was found for peak force during the MVC ($F_{12,96}$ = 0.802, P = .647). Similarly, the interaction between ratio of muscle activation for peak force was not significant for Time by Trial Order by Condition ($F_{12,95}$ = 0.651, P = .793). However, a significant time effect was found, where peak force declined significantly one minute post-TT's (P < .001), associated with a significant reduction in post-trial peak force to sEMG ratio (P < .001, **Figure 4.6**).

4.4.5 Motivation and emotion responses:

There was no significant Trial Order by Condition for success ($F_{15,55}$ = 0.552, P = .898) or intrinsic motivation ($F_{15,55}$ = 0.879, P = .510). No significant Trial Order by Condition was found in the Sports Emotion Questionnaire post TT's for: anxiety (P = .655), dejection (P = .310), excitement (P = .390), anger (P = .514) and happiness (P = .210).

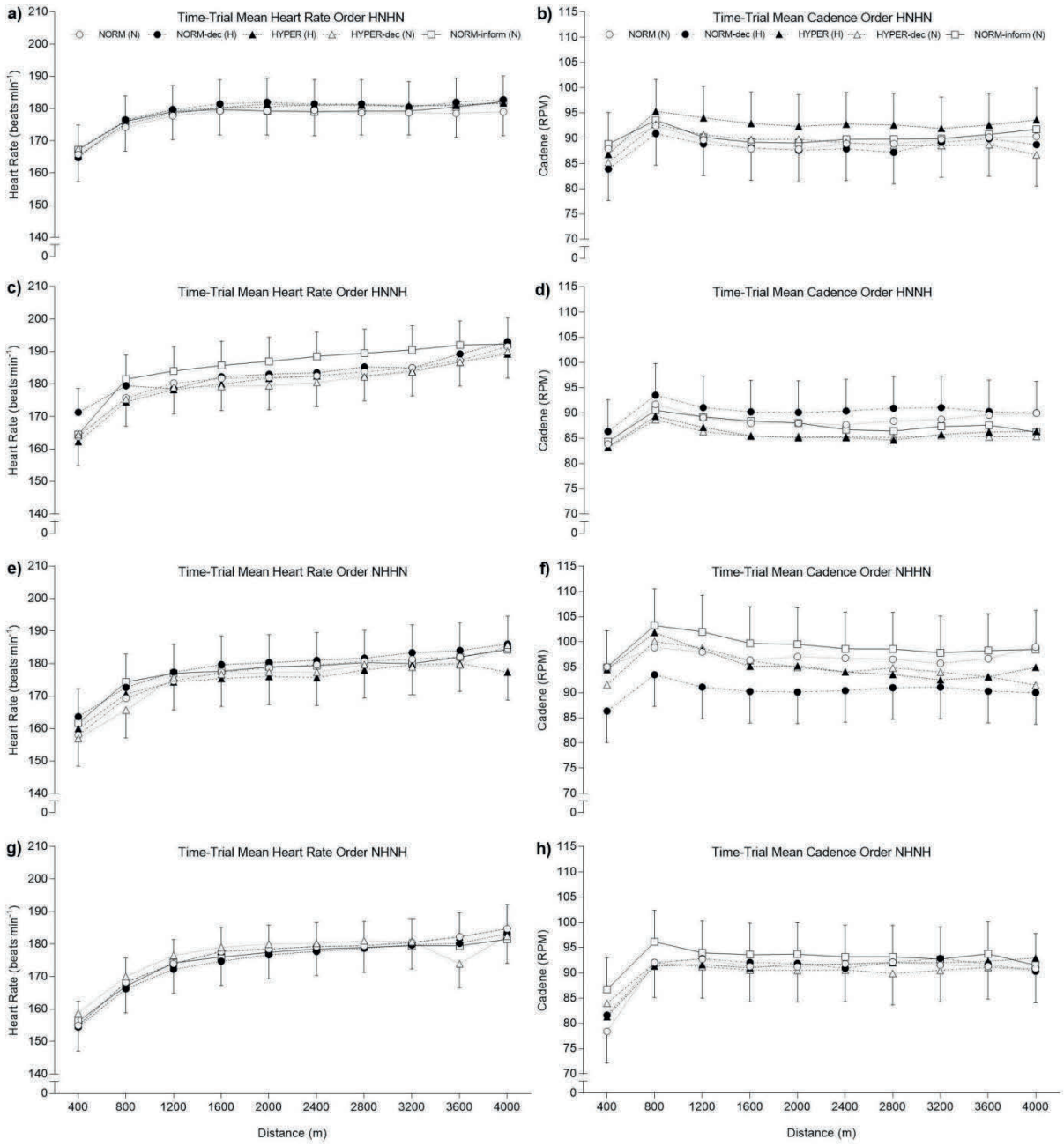


Figure 4.4 Mean heart rate responses (**left panel**) and cadence (**right panel**) at 400-m intervals for each order group during 4000-m time-trials. Results are reported as mean estimates ± 95% confidence intervals.

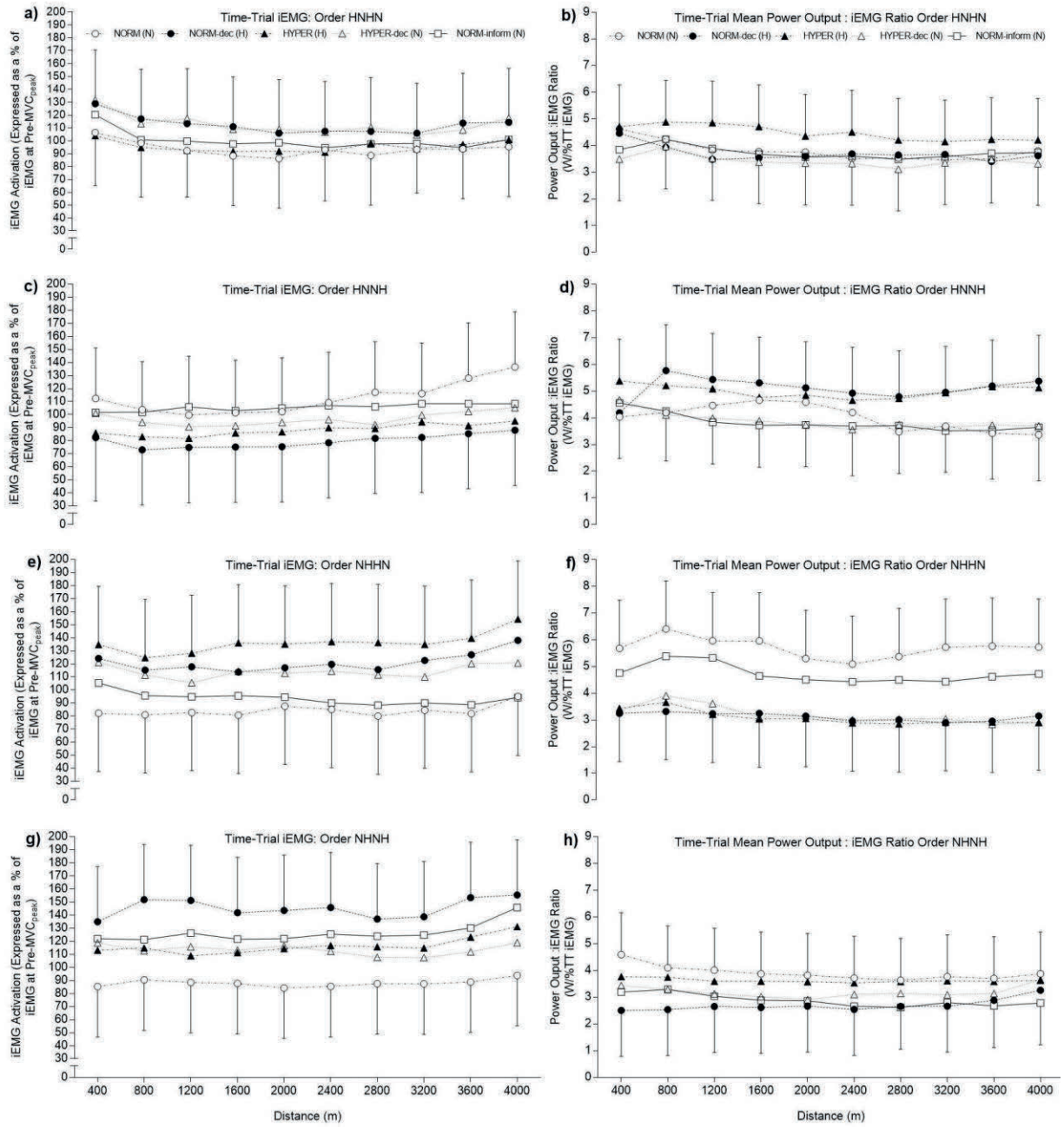


Figure 4.5 Mean integrated electromyography (iEMG) activity of the right vastus lateralis leg muscle (**left panel**), and mean ratio of power output to iEMG activity (**right panel**) at 400-m intervals for each order group during 4000-m time-trials. Results are reported as mean estimates ± 95% confidence intervals.

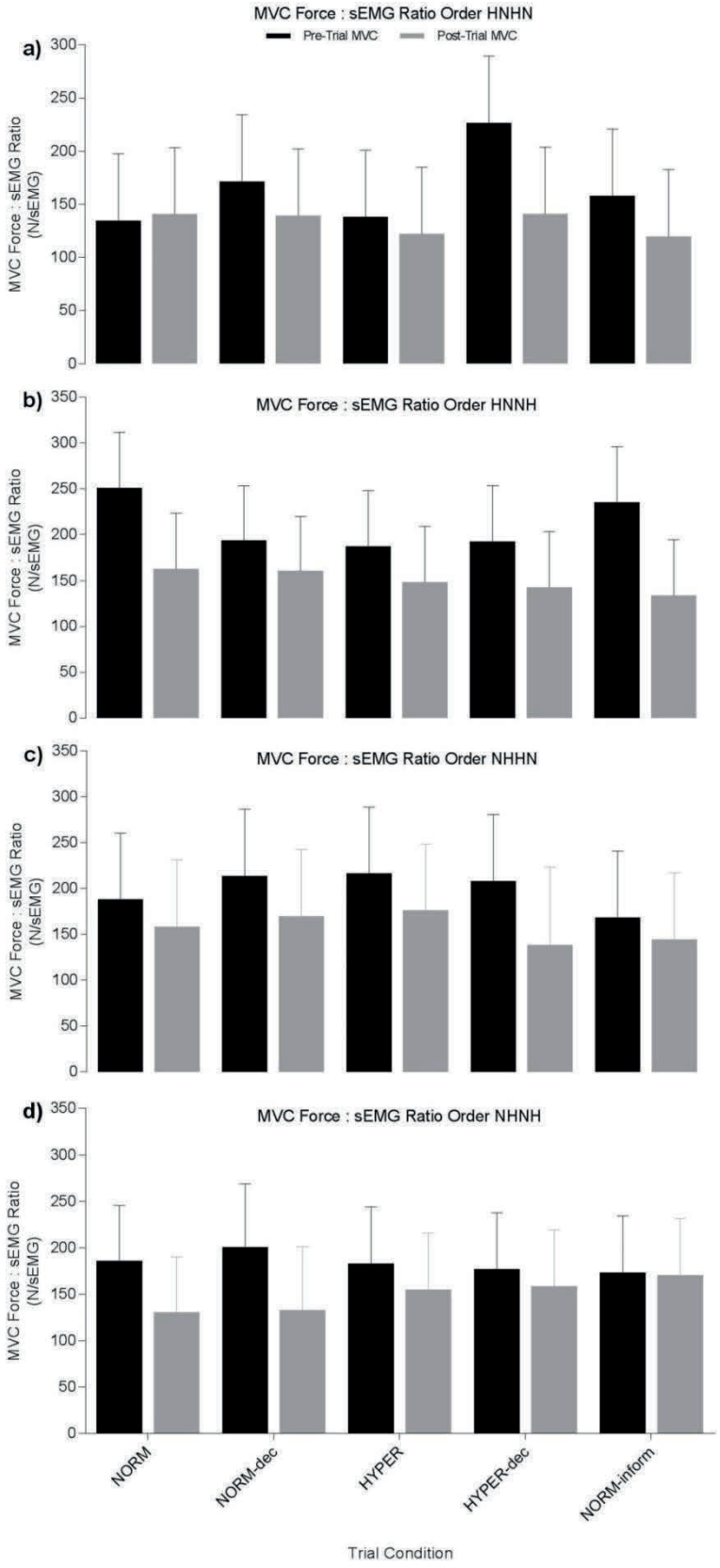


Figure 4.6 Ratio of peak force to surface electromyogram during pre- and post-trial maximal voluntary contractions. Results are reported as mean estimates ± 95% confidence intervals.

4.5 DISCUSSION:

The present study demonstrated FiO_2 content deception trials were not sufficient to improve performance in a subsequent normoxic cycling TT. However, cyclists who completed trials in HNNH did improve performance in NORM-INFORM trial by ~ 25 W (7%) or ~ 8 s (2%). In comparison, no significant change was found in NORM-INFORM compared to NORM across the other three orders. In these three trial orders, performances were within the typical error previously reported for simulated TT's completed on a cycle ergometer (< 6 W or 0.7–1.4 seconds) (170), therefore, there was no decrement in performance irrespective of trial order.

4.5.1 Exercise regulation in normoxia after oxygen deception:

Previous studies have suggested if cyclists were able to improve performance in a normoxic informed trial, it might indicate they had accessed an exercise reserve above their perceived maximal capacity (91, 158, 169). Overall, ten cyclists improved, while four declined and one had no change in performance. However, when accounting for the order of trials completed, the order appears effective in disrupting performance. Of interest, was the observed MPO improvement by ~ 7% in NORM-INFORM by cyclists completing order HNNH (**Figure 4.2c**), which was associated with a significantly greater HR response (**Figure 4.4c**) similar to earlier reports after deception was revealed (91). By comparison, previous reports have suggested athletes can access reserve capacities up to 2% for MPO before the exercise intensity becomes intolerable (169). One possible explanation is knowledge of previous trial performance, compared to perception of effort during NORM-INFORM, may have influenced cyclists belief to upregulate exercise intensity (120). However, since we found no significant change in pre-motivation or post-trial emotional responses across all trial orders this is somewhat speculative. In comparison to other trial orders, cyclists completing order

HNNH improved performance in two trials: HYPER-DEC and HYPER, prior to NORM-INFORM. Therefore, an increased belief or confidence may have been developed over this particular order leading up to NORM-INFORM. However, a measure of belief or competency was not quantified before the final trial. A further explanation for the observed improvement in NORM-INFORM might be that cyclists inspired hyperoxia in their preceding TT. Although this is unlikely as no improvement was observed in NORM-INFORM when cyclists completed trials in order NHNH. The discrepancy may be explained by the order in which trials were performed, where MPO was upregulated between 1600-m to 4000-m for all TT's in order HNNH compared to NHNH, suggesting the FiO_2 in preceding TT's may have altered pacing in subsequent trials as previously reported (34). It is possible improved performance in NORM-INFORM by those cyclists completing trial order HNNH was the result of a training effect (170). However, given performance improvements in NORM-INFORM were evident only for these participants this explanation seems unlikely. Finally, a possible explanation for a decrease or no improvement in performance for cyclists completing the other trial orders may possibly be the result of a negative experience in prior trails, whereby they over exerted themselves in a particular trial when expecting the hyperoxic gas thus implementing a more conservative approach to pacing in subsequent trials including the final NORM-INFORM trial (34).

4.5.2 Can a series of oxygen deception trials build psycho-physiological belief?

An additional aim was to examine whether environmental deception changed pacing during the deception trials themselves. Cyclists who completed order HNNH also improved MPO in HYPER-DEC by ~ 17 W (5%) compared to NORM. As a result, cyclists in HYPER-DEC were able to mitigate the limitations of inspiring normoxia when believing they were

inspiring hyperoxia, suggesting this belief influenced their performance. Therefore, it is possible improved TT performance from inspiring hyperoxia is due to physiological and potentially a psychological enhancement (placebo effect). Consequently, the order described above, may have been sufficient to disturb cyclists routine pacing template, causing the current work-rate to be upregulated despite normoxia being inspired. In comparison, cyclists maintained greater MPO in NORM-DEC compared to NORM across all orders. Therefore, believing they were inspiring normoxia did not appear to inhibit the benefits of inspiring hyperoxia, contrary to many models and theories of exercise regulation which emphasise the importance of prior experience overcoming peripheral feedback (117). This finding is in agreement with previous reports, where cyclists will adjust exercise intensity despite being unaware of inspiring hyperoxia, likely a result of associated mechanisms responding to incoming sensory feedback from the periphery (10, 78, 87, 105, 137, 187). Therefore in the present study, it is possible feedback from sensory afferents in the periphery provided a sufficient stimulus to overcome any pre-existing pacing template (10); for instance, blood chemoreceptors (137), group III-IV muscle afferents (10) or metabolic milieu (105). A novel finding for the current investigation is feedback from peripheral afferents appeared to be sufficient to overcome any pre-exercise expectations regarding achievable exercise intensity to the expected FiO_2 available.

4.5.3 Effects of exercise regulation during self-paced hyperoxic time-trials:

The current investigation supports previous studies which have found increased FiO_2 improves self-paced exercise (10, 105, 137, 187). However, irrespective of trial order, a novel finding is the benefit of inspiring hyperoxia appears not only the result of physiological responses from a change in FiO_2, per se, but also a psychological response, indicated by the subtle PO increase in HYPER compared to NORM-DEC. Increasing oxygen availability has

been attributed to an increased $\dot{V}O_2$ and faster $\dot{V}O_2$ kinetics (137), increased oxygen availability at skeletal muscles and reduced biochemical and physiological disturbances to homeostasis (187). For instance, inspiring hyperoxia increases cerebral oxygenation and reduces levels of fatiguing metabolites at working musculature (105), often limiting factors for exercise in normoxia (187). Furthermore, other factors, such as the effectiveness of respiratory muscles to uptake oxygen, muscle recruitment and teleoanticipation may also affect exercise intensity in hyperoxia before limiting levels of fatigue are reached (10, 105, 137, 187).

4.5.4 Voluntary force production and muscle activation:

Differences in muscle activity may be attributed to changes in either the central drive in motor unit recruitment or the frequency rate of motor units at the measured working musculature (70). We found consistent changes in muscle activation, regardless of trial order, condition or segment. In addition, the magnitude of force reduction in post-trial MVCs was similar across all conditions, which suggests hyperoxia did not decrease the development of peripheral muscle fatigue (187). However, exploratory graphical inspection of iEMG signals suggests some differences in muscle activity between trials. It is possible oxygen content, deception and trial order completed, may have increased uncertainty within the trial by altering pre-trial knowledge, expectations and subsequently central motor drive which may explain the varied muscle activities. However, we acknowledge there is inherent measurement variability when sampling iEMG activity during dynamic exercise, often resulting in interpretation difficulties (170). Nevertheless, changes in iEMG amplitude can still provide a practical reflection of changes in muscle activation during self-paced exercise (187).

4.6 PRACTICAL APPLICATIONS:

- Hyperoxic and oxygen deception TT's have the potential to build belief and improve performance in subsequent normoxic trials or competitive events.
- The order in which hyperoxic and deception TT's are performed may be used to encourage athletes to deviate from their routine pacing template.
- An appropriate pacing strategy should be considered when the FiO_2 is altered in training or competition to avoid misjudging pacing which may have positive or negative consequences on performance.
- Future studies manipulating the FiO_2 should consider the effects trial order and participants knowledge of the FiO_2 may have on exercise regulation.

4.7 CONCLUSION:

A series of oxygen deception trials did not improve performance in a subsequent, normoxic TT, once the deception had been revealed, despite improvements being observed in prior hyperoxic and normoxic-deception trials. However, the order trials were completed appears to have influenced performance, as one order performance improved in the NORM-INFORM compared to NORM.

5.0 STUDY THREE: NEGATIVE PERFORMANCE DECEPTION ALTERS PACING AND PERFORMANCE DURING A 4000-m HYPEROXIC CYCLING TIME-TRIAL.

Citation: **Davies, M. J**., Clark, B., Garvican-Lewis, L. A., Welvaert, M., Gore, C. J., & Thompson, K. G. (2018). Negative performance deception alters pacing and performance during a 4000-m hyperoxic cycling time-trial.

5.i FORM E: DECLARATION OF CO-AUTHORED PUBLICATION CHAPTER

For use in theses which include publications. This declaration must be completed for each co-authored publication and to be placed at the start of the thesis chapter in which the publication appears.

Declaration for Thesis Chapter 5

Declaration by candidate

In the case of Chapter 3 the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Study design, data collection and analysis and study write up	65

The following co-authors contributed to the work.

Name	**Nature of contribution**	**Extent of contribution (%)**	**Contributor is also a student at UC Y/N**
Bradley Clark	Assistance with study design, data collection, analysis and interpretation of results and proof reading of drafts	10	N
Laura Garvican-Lewis	Assistance with study design, interpretation of results and proof reading of drafts	5	N
Marijke Welvaert	Assistance with study design, analysis and interpretation of results and proof reading of drafts	5	N
Chris Gore	Assistance with study design, interpretation of results and proof reading of drafts	5	N
Kevin Thompson	Assistance with study design, analysis and interpretation of results and proof reading of drafts	10	N

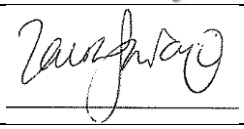
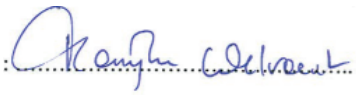
Candidate's Signature		**Date** 24/04/2018

Declaration by co-authors
The undersigned hereby certify that:

(13) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors.
(14) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
(15) they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
(16) there are no other authors of the publication according to these criteria;
(17) potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit; and
(18) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s)	Australian Institute of Sport Department of Physiology, Bruce, ACT Research Institute for Sport and Exercise, University of Canberra, Bruce ACT

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant.]

Co-author Signature Number	**Co-author's Signatures**	**Date**
Signature 1		24/04/2018
Signature 2		24/04/2018
Signature 3		24/04/2018
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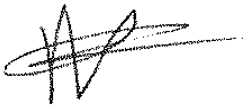
Signature 5		24/04/2018

5.1 ABSRACT

Background: We investigated whether negative deceptive feedback could be used to alter performance in a hyperoxic time-trial (TT) and if any performance changes would be sustained in a subsequent normoxic trial.

Method: Twenty-three male cyclists (mean ± SD: age 33.8 ± 8.6 years; stature 180 ± 6.1 cm; body-mass 73.3 ± 7.7 kg; peak oxygen uptake 63 ± 7 $mL \cdot kg^{-1} \cdot min^{-1}$; maximal aerobic power 4.9 ± 0.6 $W \cdot kg^{-1}$) completed seven, 4000-m cycling TT's. In two familiarisation, self-paced TT's, cyclists inspired normoxic air (NORM; fraction of inspired oxygen (FiO_2): 0.21 ± 0.04). In trials three and four hyperoxic air (FiO_2: 0.36 ± 0.03) was inspired, with trial three self-paced (HYPER), whereas in trial four (HYPER-PACER), cyclists raced an on-screen avatar, representing the mean power output (MPO) from HYPER. In trial five, cyclist's completed a self-paced TT in normoxia (NORM-POST). For trial six, (HYPER-PACER-DEC102%), cyclists inspired hyperoxic air and were deceived of the 2% MPO increase to the avatar above HYPER-PACER. In the final TT (NORM-PACER104%), deception was revealed, and cyclists raced an avatar at a MPO 4% above NORM and were accurately informed.

Results: Performance (MPO and elapsed time) significantly improved in HYPER-PACER-DEC102% compared to HYPER-PACER ($P < 0.001$), and in NORM-PACER104% compared to NORM ($P < 0.001$).

Conclusions: Performance improvements were associated with varying changes to anaerobic-aerobic energy contributions. In summary, a performance reserve may exist in hyperoxia, while additionally it appears the benefits of the hyperoxic trials were sustained in the subsequent, normoxic trials.

Keywords: pacing, exercise reserve, hyperoxia, deception, feedback

5.2 INTRODUCTION

During self-paced exercise, athletes adopt a pacing "strategy" to regulate exercise intensity, often believed to be the product of a pre-determined 'template' built on prior exercise experience (3). Once the exercise task begins, internal and external feedback is integrated to determine if the current work-rate should be maintained or adjusted (52). Recently, research has demonstrated manipulation of an athlete's task expectations and beliefs can allow athletes to access an exercise reserve and improve performance (89, 91, 158, 169). The most common form of task manipulation is the non-invasive provision of deceptive information regarding exercise performance or the task environment (88, 90). In general, deception methods attempt to create a higher level of task uncertainty before or during performance to encourage athletes to deviate from their routine strategy (204).

To challenge athletes task expectations or goals, researchers have altered task feedback such that athletes believe they are performing better (positive) or worse (negative) than reality (40). Recent evidence suggests negative performance deception is the most effective method to elicit performance improvements, both during deception trials themselves, and in a subsequent trial after deception is revealed (40). Negative deception is typically achieved via misrepresentation of a previous performance trial in the form of an on-screen avatar, which is programmed, unknowingly to the athlete, above previous best performances (i.e. 2% greater) (40, 89, 91, 158, 169, 203). Performance improvements associated with negative deception trials are believed to be the result of athletes accessing an exercise reserve. The exercise reserve is defined as the remaining metabolic energy that has not been used once a self-paced task has been completed, and is presumably maintained to prevent catastrophic performance failure (173). However, despite recent advancements many uncertainties remain including,

the upper limit of the reserve, how improvements manifest (i.e. via anaerobic or aerobic energy pathways) and the techniques available to use the reserve during competition.

Another method to change task expectation and routine pacing patterns is to alter the inspired oxygen content. Previous research has demonstrated that when athletes were blinded to the fraction of inspired oxygen (FiO_2), hyperoxia: ($FiO_2 > 0.21$) is sufficient to acutely improve self-paced TT performance by ~ 6% (110). Numerous mechanisms may explain this improvement, however it is most likely related to an increased availability of oxygen at the working musculature and subsequent reduction in physiological disturbances to homeostasis (187). Interestingly, despite supplemental oxygen permitting a greater exercise capacity (10), it remains to be elucidated whether an exercise reserve exists in hyperoxia. Previous observations in normoxia TT's suggest finite anaerobic energy supplies, normally left in reserve, can be accessed when athletes are given performance deceptive feedback TT's (158, 169, 173). Therefore, if an exercise reserve exists and can be used in hyperoxia, it may provide additional insight into the physiological source of the energy reserve (89, 91, 158, 169), however, whether an energy reserve exists in hyperoxia remains unknown. Moreover, whether the combination of hyperoxia and performance deception can improve subsequent performance once deception is revealed warrants further investigation.

Thus, the primary aims of this study were to: i) investigate if an exercise reserve exists in hyperoxia and ii) whether negative performance feedback could be used to change task expectations and build psycho-physiological belief to alter pacing to improve performance in normoxia. Collectively, this information will further our understanding of how hyperoxia and negative performance deceptive information alters trained athletes exercise regulation and determine if these techniques could be used in training to enhance race performance.

5.3 METHODS

5.3.1 Study Design:

Cyclists completed seven, 4000-m cycling TT's in a laboratory and in all trials were correctly informed of the FiO_2 (Figure 5.1). Briefly, cyclists first completed a graded exercise test (GXT) to exhaustion and two baseline trials while inspiring normoxic air (NORM; FiO_2: 0.21 ± 0.04). In trial three (HYPER) and four (HYPER-PACER) cyclists inspired hyperoxic air (FiO_2: 0.36 ± 0.03). Trial three was self-paced while in trial four, cyclists raced a virtal on-screen avatar, which accuractely represented mean power output (MPO) from trial three. In trial five cyclist's completed a self-paced TT (no pacer) in normoxia (NORM-POST; FiO_2: 0.21 ± 0.04) to evaluate how previous hyperoxic trials may influence pacing and normoxic performance. For trial six (HYPER-PACER-DEC102%; FiO_2: 0.36 ± 0.03), cyclists inspired hyperoxic air and were misinformed that the avatar was set at their previous best MPO when in reality it was set at 2% above their previous best MPO. Deceptive manipulation of the avatar by 2% was chosen based on earlier work suggesting a 2% increase in MPO is subtle enough to improve performance while minimising detection (39). On arrival for their seventh and final testing session (NORM-PACER104%; FiO_2: 0.21 ± 0.04), the primary researcher revealed the deception in HYPER-PACER-DEC102%, and cyclists were asked to complete a final normoxic trial while racing against an avatar set 4% above NORM (NORM-PACER104%). A 4% increase to the avatar's MPO was chosen to replicate a hyperoxic TT when cyclists inspired normoxic air, based on the mean improvement to performance determined in Study Two when cyclists inspired hyperoxia. In the final trial, cyclists were accurately informed of the increased pacer MPO above their best previous normoxic trial. All tests were completed over a two week period, separated by at least 48 hours and completed on a VelotronDynafit Pro cycle ergometer (RacerMateInc, CITY, WA, USA), fitted with an

SRM power meter (Ingenieurbüro Schoberer, Jülich, Germany) which sampled PO at 1Hz, calibrated using a dynamic calibration rig as previously described (2) [Appendix 3]. The ergometer was paired with the Velotron 3D software (RacerMateInc, CITY, WA, USA). Cyclists were instructed to maintain their normal diet, refrain from strenuous exercise, caffeine or alcohol in the 24 hours each testing session.

5.3.2 Participants:

Twenty-three trained male cyclists (mean ± SD: age 33.8 ± 8.6 years; stature 180 ± 6.1 cm; body-mass 73.3 ± 7.7 kg; peak oxygen uptake ($\dot{V}O_{2peak}$) 63 ± 7 $mL \cdot kg^{-1} \cdot min^{-1}$; maximal aerobic power (MAP); 4.9 ± 0.6 ($W \cdot kg^{-1}$) volunteered to take part in the study. All cyclists provided informed consent before participating. The study was approved by the University of Canberra Research Ethics Committee. Finally, participants received a one-page factsheet before participating and were briefed throughout the study on the ergogenic benefits of inspiring hyperoxia on performance [Appendix 2].

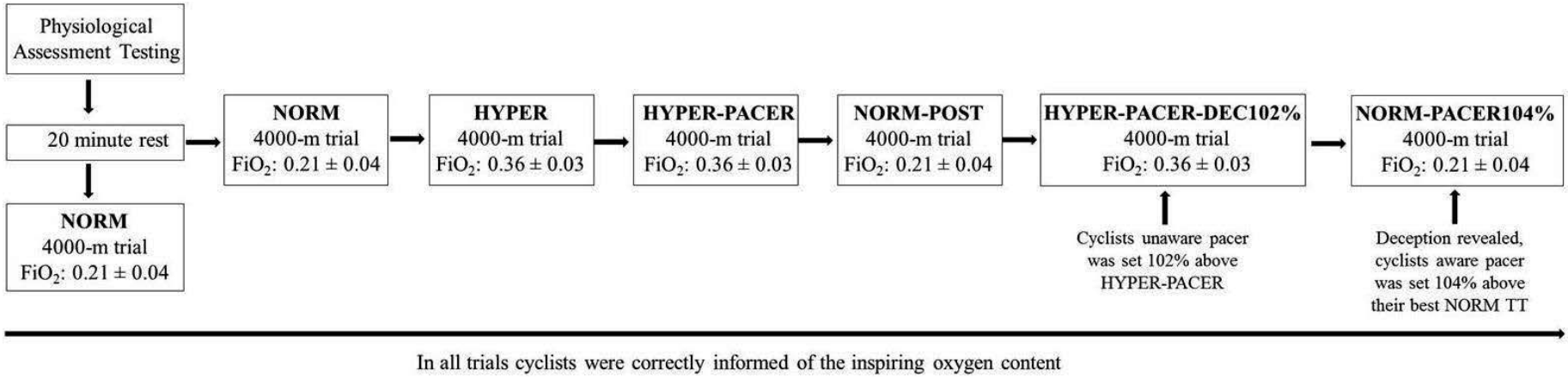


Figure 5.1 Study design, including inspired FiO_2 and information provided to cyclists for each trial.

5.3.3 Experimental Procedures

Preliminary Testing:

Prior to experimental trials, cyclists completed a GXT for determination of $\dot{V}O_{2peak}$ and MAP. After a 10-minute self-paced warm-up, PO was set at 150 W and increased by 25 W every three minutes until volitional exhaustion or when cadence dropped below 80 revolutions per minute (RPM). Respiratory gases were collected by the ParvoMedics TrueOne 2400 system (ParvoMedics, Salt Lake City UT, USA) and $\dot{V}O_{2peak}$ defined as the highest $\dot{V}O_2$ over a 30 s period. The ParvoMedics system was validated prior to the study using a metabolic calibrator (73) which has been described previously (39, 107). MAP was determined as the final completed stage plus the proportion of any uncompleted stage attempted during the GXT (96). Heart rate (HR) and arterial (pulse) oxygen saturation (S_pO_2) were recorded continuously during trials using a portable telemetry device (Polar, model s810i, Kempele, Finland) and finger pulse oximeter (Onyx Vantage, model 3150, Nonin, Minnesota, USA), respectively. Before each test, the ergometer was adjusted to replicate each cyclist's own racing bicycle, including crank length, handlebars, saddle and pedals. These measurements remained consistent for subsequent trials.

4000-m time-trial protocol:

Before each TT, cyclists were seated for 10 minutes to complete a 'wash in' phase where they inspired the gas mixture used in the subsequent trial. The purpose of the wash in phase was to stabilise homeostatic disturbances and alterations to regulatory processes influenced by sensory afferents in the periphery detecting changes in the FiO_2 (185). Cyclists then completed an 11-minute standardised warm-up. Briefly, cyclists completed four minutes at 60% MAP, three minutes at 70% MAP, two minutes at 80% MAP, one minute building from

80-90% MAP, a six second sprint, finishing with one minute of active recovery (113). Following the last stage of the warm-up cyclists had one minute of passive recovery before commencing the TT. Before each trial the primary researcher instructed cyclists to give a maximal effort and complete the TT as fast as possible. Performance time, PO, cadence, HR and S_pO_2 were recorded at one second intervals, and rating of perceived exertion (RPE) was recorded at each kilometre using Borg's 6–20 scale (26). Data were assigned to segments representing 5% of the distance completed for each trial, excluding HR and RPE which were assigned to 10% and 25% segments, respectively. To describe the variability in the pacing strategy between trials we also calculated the pacing index using the following equation (208).

$$(\textit{Segment mean power output} / \textit{Overall trial mean power output}) * 100$$

Constant visual feedback was given regarding distance travelled and gear selection via a large television screen paired with the cycling ergometer, providing a visual representation of cyclists completing a virtual 4000-m course. In addition, instantaneous S_pO_2 was displayed on a computer monitor to accurately inform cyclists they were not being deceived of the inspired oxygen content. During competitive TT's, cyclists raced a virtual on-screen pacer set at or above MPO from a previous trial using the Velotron software as previously described (169). However, all other feedback including elapsed time, PO, velocity, cadence and HR was withheld. Cyclists were free to self-select cadence, gear ratio and vary cycling posture (e.g. seated or standing) throughout. Cooling was provided by a fan placed one meter in front of cyclists (~20-km.h^{-1}, laboratory temperature 21.6 ± 1.1°C). Finally, verbal encouragement was given by the primary researcher from a pre-prepared script at 500-m intervals and at 250-m and 100-m to completion (74).

Environmental manipulation:

For trials three, four and six cyclists inspired hyperoxic air. To achieve this, the primary researcher filled two large aluminized mylar bags (1200 L) with hyperoxic air, which was produced by mixing normal room air with medical grade oxygen (O_2 100%, BOC Gases, Canberra, Australia), or ambient air for normoxic trials. Both bags were filled simultaneously to the desired volume and gas content sampled before each trial by a paramagnetic oxygen analyser and infrared carbon dioxide analyser (ParvoMedics, Salt Lake City UT, USA, normoxia actual: FiO_2 0.21 ± 0.04; hyperoxia actual: 0.36 ± 0.06). The level of hyperoxia was selected based on available literature where a subtle increase in the inspired oxygen content (0.35 – 0.40 FiO_2) can improve performance but be difficult for participants to detect when blinded (163). In addition, the administered hyperoxic air was based on recommendations from the Australian Institute of Sport (AIS) Medical Team on safe and ethical use of hyperoxic gas.

Gas analysis during performance trials:

Inspired gas was delivered via a Hans Rudolph one-way valve (Hans Rudolph Inc, Shawnee KS, USA), with a secondary one metre tube directing expired air to the ParvoMedics system. During hyperoxic trials gas sampling was achieved by setting the ParvoMedics system to *'hyperoxic mode'*. To validate the ability of the system to accurately measure the inspired and expired gas content, the metabolic system was checked using a bottle of alpha gases (O_2 40.0%, CO_2 5.9%, balance N, BOC Gases, Canberra, Australia). In addition, aerobic (P_{AER}) and anaerobic (P_{AN}) contributions to total PO were calculated according to the methods previously described (43), using gross efficiency (GE) estimated at 60% MAP for four minutes during the warm-up.

Gross efficiency was calculated using the following equation:

$$GE = (PO / \text{metabolic power input}) \times 100\%$$

The metabolic power input was calculated by multiplying $\dot{V}O_2$ with the oxygen equivalent in the following equation as suggested by Garby and Astrup (64).

$$\text{Metabolic power input} = \frac{\dot{V}O2}{60} (4940 \times \text{respiratory exchange ratio} + 16040)$$

To determine GE oxygen consumption had to be in steady-state with a respiratory exchange ratio < 1.0 to exclude the contribution of unmeasured anaerobic work described by van Ingen Schenau (191). Finally, P_{AER} and P_{AN} contributions to total PO were calculated by multiplying metabolic power input with GE for each 15-second interval of the trial.

Electromyography and maximal voluntary contraction:

Five minutes before beginning the 'wash in' phase, cyclists performed three, five second maximal voluntary contractions (MVC) as described previously (189). Briefly, after a three-minute warm-up of easy pedalling, cyclists completed MVCs of the right leg extensors, on a 30 s cycle, at zero, 30 and 60 s. The same procedure was repeated one minute after each TT. Before each contraction cyclists were instructed to "push" against the ankle strap as hard as possible to provide an external focus, previously suggested to elicit greater total force production (74). Surface electromyography (sEMG) from cyclists right vastus lateralis leg muscle was recorded during all MVCs and continuously during each TT. The skin over the muscle belly was shaved, lightly abraded and cleaned, before the placement of two electrodes positioned on the midline belly of the muscle, inter-electrode distance of 30-mm (33). An electronic goniometer (ADInstruments, NSW, Australia) was positioned and secured on the lateral side of the right knee to set a 100° angle of knee flexion in the pre- and post-MVC, and secondly to trigger the corresponding sEMG at 100° knee flexion (99.7 ± 0.1°) during each downward pedal stroke during TT's. Raw sEMG and goniometer signals were sampled

at 2,000-Hz via LabChart v8.1 (ADInstruments, NSW, Australia) system, filtered, rectified and smoothed to produce a linear envelope (or integrated EMG, iEMG), as described previously (33). Thus, cycling sEMG data were amplitude-normalised, using the TT peak iEMG value at 100° knee flexion, normalised to the greatest peak iEMG value associated with the highest force produced in the pre-MVCs.

Psychological questionnaires:

Cyclists completed the motivational component of the Dundee Stress State Questionnaire (DSSQ), to measure success motivation (motivation to excel in performance) and intrinsic motivation (cyclists interest in the trial content) before each trial (115). In addition, a Sports Emotion Questionnaire (SEQ) was completed after each trial as a sport-specific measure of post-competitive emotion to assess: anger, anxiety, dejection, excitement and happiness (92).

5.3.4 Statistical Analysis

Data were analysed with a General Linear Mixed Model which included fixed effects for trial order, condition and segment. All models were estimated using Restricted Maximum Likelihood as implemented in the R package lme4 (21, 179). A random intercept for Subjects was included to account for intra-individual dependencies and inter-individual heterogeneity. Model assumptions were assessed through visual inspection of residual plots revealing no obvious deviations from homoscedasticity. However, heavier tails than would be expected were found under the normal distribution. To account for this, bootstrapped confidence intervals were obtained to minimize distributional impact. P-values were obtained using Type II Wald F tests with Kenward-Roger degrees of freedom as implemented in the R package car (62). Results are reported as mean estimates and 95% confidence intervals and a significance level of $p < 0.05$ was chosen a priori for all tests.

5.4 RESULTS

5.4.1 Time-trial performance and pacing:

There was no significant Condition by Segment interaction for TT MPO ($F_{114,2878} = 0.95$, $P = 0.65$). However, significant Condition ($F_{6,2878} = 120.32$, $P < 0.001$) and Segment ($F_{19,2878} = 117.12$, $P < 0.001$) main effects were found for MPO (**Figure 5.2a**). Mean power output was significantly higher in HYPER-PACER-DEC102% compared with HYPER-PACER (392 W, 95% CI = [374, 410] vs. 384 W [366, 402]), which led to a concomitant improvement in performance time by ~ 3 s (HYPER-PACER-DEC102%: 340 s [336, 344] vs. HYPER-PACER: 343 s [339, 347], $P < 0.001$). Similarly, MPO was significantly higher in NORM-PACER104% compared with NORM (367 W [349, 386] vs. 349 W [331, 367]), leading to improved performance time by ~ 7 s (NORM-PACER104%: 349 s [345, 353] vs. NORM: 356 s [352, 360], $P < 0.001$). Performance was significantly improved in a self-paced, hyperoxic trial (HYPER: 379 W [361, 397]) compared to NORM (349 W [331, 367]). Additionally, MPO was significantly increased in a normoxic trial after two preceding hyperoxic trials compared to NORM (NORM-POST: 356 W [337, 374]). However, adding a competitor while inspiring hyperoxic air (HYPER-PACER: 384 W [366, 402]) resulted in no significant difference compared to no pacer (HYPER: 379 W [361, 397]).

A significant Condition by Segment interaction was found for the pacing index ($F_{114,2878} = 1.26$, $P = 0.04$). Specifically, the pacing index was upregulated in NORM-PACER104% and HYPER from 600-m to 1000-m compared to NORM ($P < 0.001$, **Figure 5.2b**). In contrast, post-hoc analysis of the pacing index was not different in other trials ($P > 0.05$).

The interaction between Condition by Segment for cadence was not significant ($F_{114,2878}$ = 0.29, P = 1.00), although significant main effects were found for Condition ($F_{6,2878}$ = 27.07, P < 0.001) and Segment ($F_{19,2878}$ = 9.45, P < 0.001). Specifically, cadence was significantly greater from 400-m to 1000-m in NORM-PACER104% compared to NORM (**Figure 5.2c**).

Table 5.1 Mean time to completion and mean power output during 4000-m time-trials. Results are reported as mean estimates ± 95% confidence intervals unless specified (mean ± standard deviation (SD)).

	NORM	**HYPER**	**HYPER+PACER**	**NORM-POST**	**HYPER102%**	**NORM104%**
FiO_2 (M ± SD)	20.94 ± 0.04	35.98 ± 0.57	35.98 ± 0.57	20.94 ± 0.04	35.98 ± 0.57	20.94 ± 0.04
Time to completion (sec)	356 (352–366)	345 (341–349)	343 (339–347)	352 (348–357)	340 (336–344)	349 (345–353)
MPO (W)	349 (299–499)	379 (335–505)	384 (338–491)	356 (304–510)	392 (346–508)	367 (319–510)

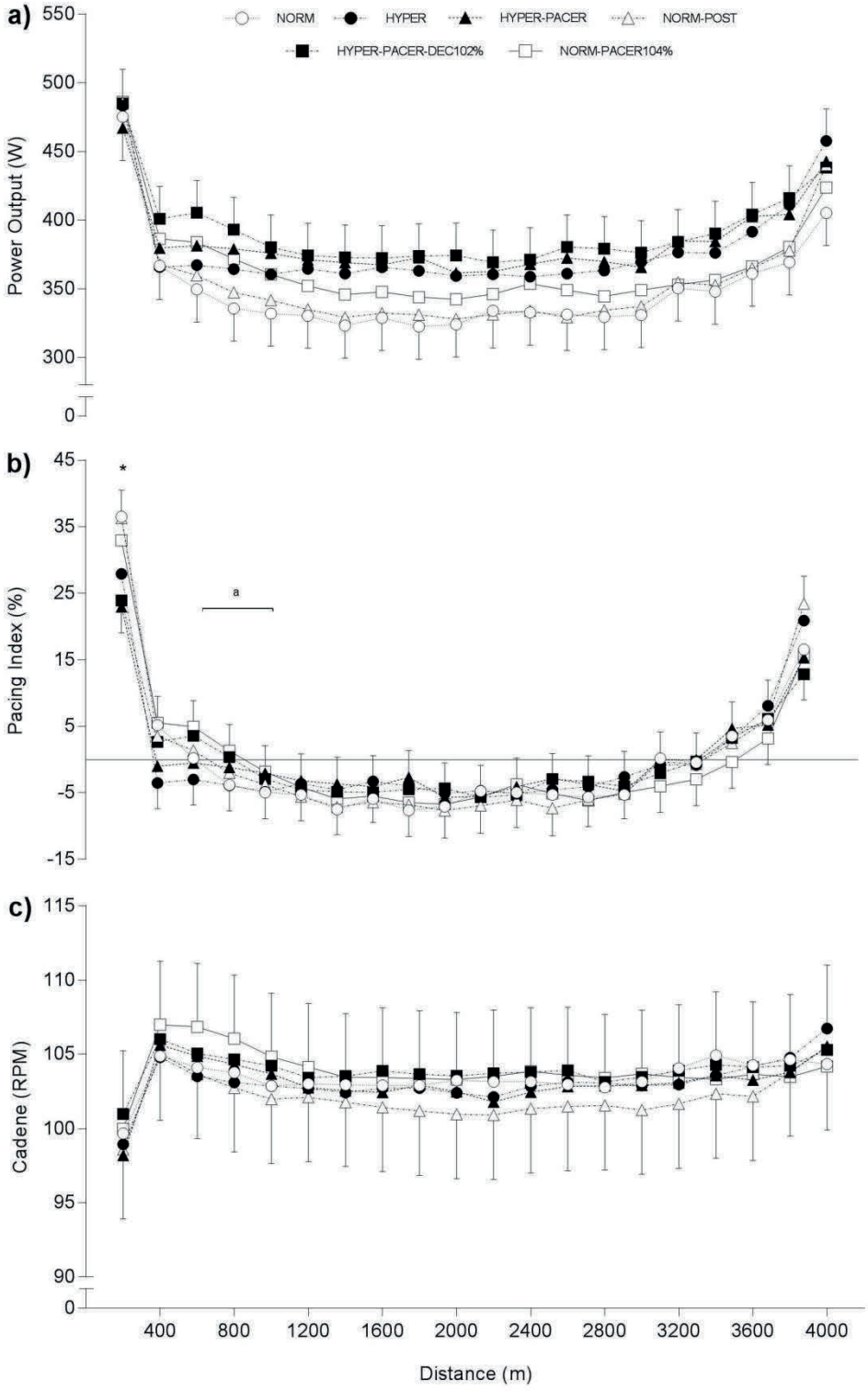


Figure 5.2 Mean power output (**a**) and associated pacing index (**b**) and cadence (**c**) at 200-m intervals during 4000-m time-trials. Results are reported as mean estimates ± 95% confidence intervals. a Significant difference in NORM-PACER104% compared to NORM.

5.4.2 Physiological responses:

No significant Condition by Segment interaction was found for minute ventilation ($F_{114,2065}$ = 0.57, P = 1.00), however, there was a significant main effect of Condition ($F_{6,2065}$ = 89.51, P < 0.001, **Figure 5.3a**). Specifically, ventilation was significantly higher during NORM-PACER104% (156 L·min^{-1} [144, 168]) compared to NORM (148 L·min^{-1} [136, 160]). In comparison, a significantly lower ventilation was found in HYPER compared to NORM (142 L·min^{-1} [130, 153] vs. 148 L·min^{-1} [136, 160]).

There was a significant Condition by Segment interaction for oxygen uptake ($F_{114,2065}$ = 1.51, P < 0.001), with a higher $\dot{V}O_2$ during hyperoxic trials (**Figure 5.3b**). Oxygen uptake was similar in all conditions during the first 1000-m, however, $\dot{V}O_2$ continued to increase in hyperoxic trials thereafter. Specifically, $\dot{V}O_2$ increased in HYPER from 1600-m to 4000-m compared to NORM. In addition, there was a ~ 2% increase in $\dot{V}O_2$ in NORM-PACER104% after the first 800-m compared to other normoxic TT's. A significant Condition by Segment interaction was found for P_{AER} ($F_{114,2065}$ = 1.46, P < 0.00; **Figure 5.3c**) and P_{AN} ($F_{114,2065}$ = 1.61, P < 0.001; **Figure 5.3d**) energy contributions. Specifically, in HYPER there was a significantly higher P_{AER} contribution from 1600-m to the finish compared to NORM. A higher P_{AER} contribution throughout NORM-PACER104% was found compared to self-paced normoxic TT's. In addition, there was a higher P_{AN} contribution for NORM-PACER104% in the first 2000-m compared to NORM. However, no significant difference in P_{AN} contribution was found between HYPER-PACER-DEC102% and HYPER-PACER.

There was no significant Condition by Segment interaction for HR ($F_{54,1486}$ = 0.55, P = 1.00, **Figure 5.4a**). However, significant differences were found between conditions for HR ($F_{6,1486}$ = 33.39, P < 0.001). Specifically, NORM-PACER104% produced significantly higher HRs

compared to NORM (178 bpm [173, 182] vs. 175 bpm [171, 179]). There was a significant Condition by Segment interaction for S_pO_2 ($F_{54,1477} = 2.35$, $P < 0.001$), where higher blood oxygen saturation was maintained in hyperoxic compared to normoxic TT's (**Figure 5.4b**). In addition, not only was S_pO_2 lower in normoxia, it decreased throughout the TT while remaining relatively stable throughout hyperoxic trials.

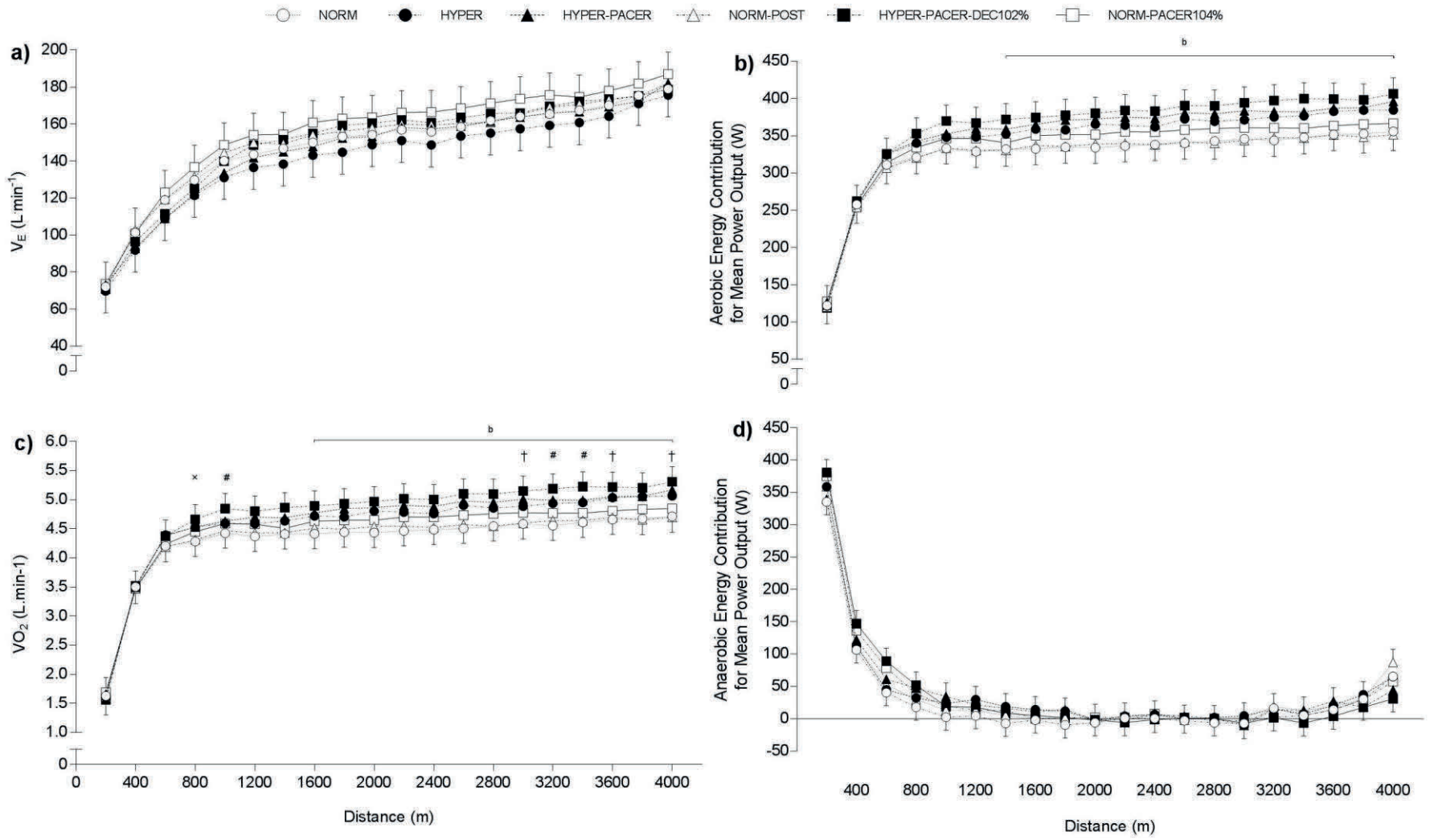


Figure 5.3 Ventilation (VE) (**a**), aerobic contribution to power output (**b**) $\dot{V}O_2$ (**c**), anaerobic (**d**) contribution to power output at 200-m intervals for each condition during 4000-m time-trials. Results are reported as mean estimates ± 95% confidence intervals. [b] Significant difference in HYPER compared to NORM *Significant difference in NORM compared to NORM-POST. #Significant difference between HYPER-PACER-DEC102% and HYPER-PACER. † Significant difference between NORM-PACER104% and NORM. [×] Significant difference in HYPER compared to NORM

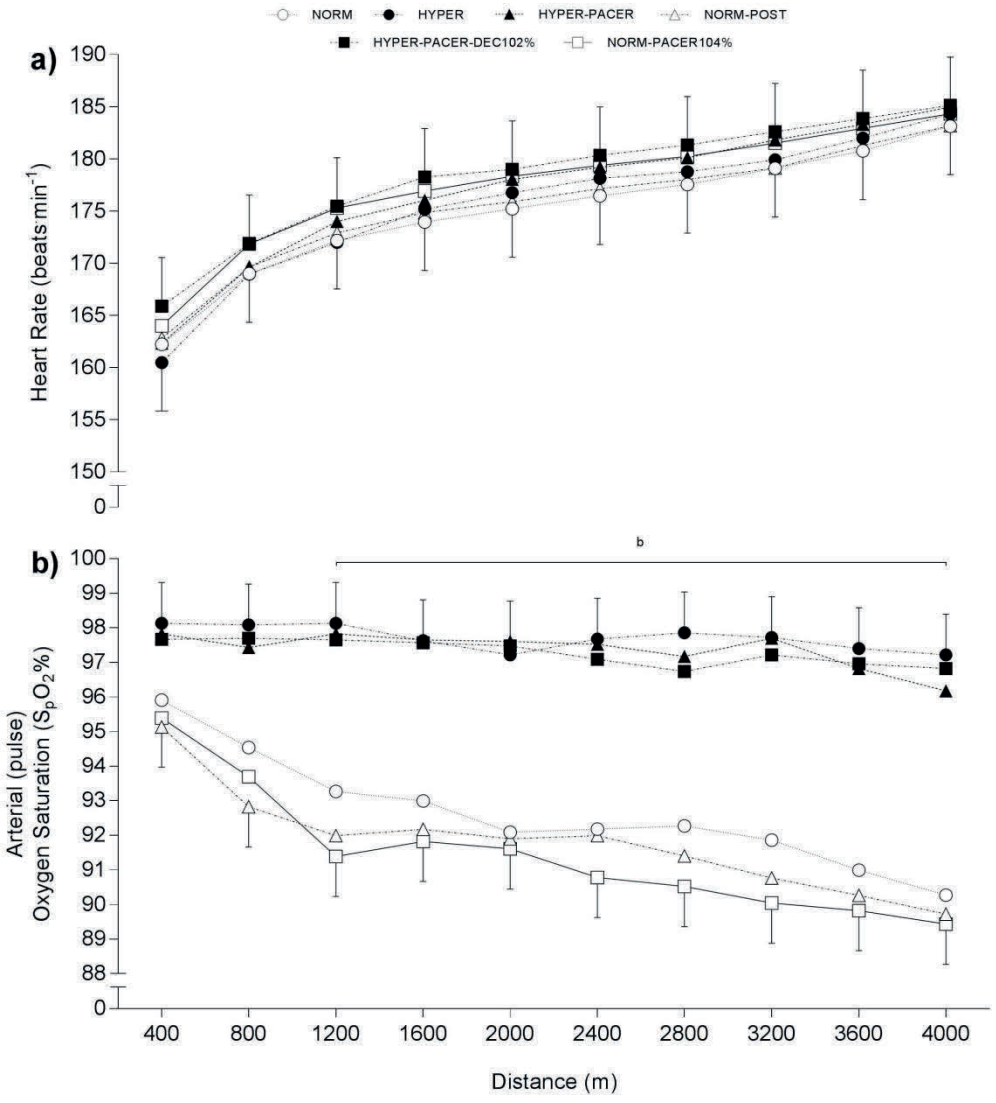


Figure 5.4 Mean heart rate (**a**) and arterial (pulse) oxygen saturation (**b**) responses at 400-m intervals for each condition during 4000-m time-trials. Results are reported as mean estimates ± 95% confidence intervals. b Significant difference in HYPER compared to NORM.

5.4.3 Muscle activation during time-trials:

There was no significant Condition by Segment interaction for muscle activation ($F_{114,2717}$ = 0.14, P = 1.00). Similarly, when comparing the ratio of muscle activation for PO, no interaction for Condition by Segment was found ($F_{114,2616}$ = 0.18, P = 1.00). In contrast, there were significant Condition ($F_{6,2718}$ = 12.25, P < 0.001) and Segment ($F_{19,2717}$ = 3.49, p < 0.001) main effects for absolute muscle activation (Figure 5a). Specifically, in HYPER-PACER-DEC102% iEMG activity (124 % [110, 139]) was significantly greater compared to normoxic trials (NORM: 108 % [93, 122]; NORM-POST: 114 % [100, 129]; NORM-PACER104%: 106 % [91, 120]). However, the ratio of muscle activation to PO was similar across all conditions (**Figure 5.5c**).

5.4.4 Voluntary force production and muscle activation:

No significant Condition by Time interaction was found for peak force during the five second MVC ($F_{6,271}$ = 0.19, P = 0.98). Similarly, when comparing the ratio of muscle activation for peak force, no interaction for Condition by Time was found ($F_{6,271}$ = 0.46, P = 0.84). However, a significant time effect was found, where peak force ($F_{1,271}$ = 65.57, P < 0.001) and muscle activation ($F_{1,270}$ = 10.93, P = 0.08) declined significantly from baseline to one-minute post-TT's (**Figure 5.5d**).

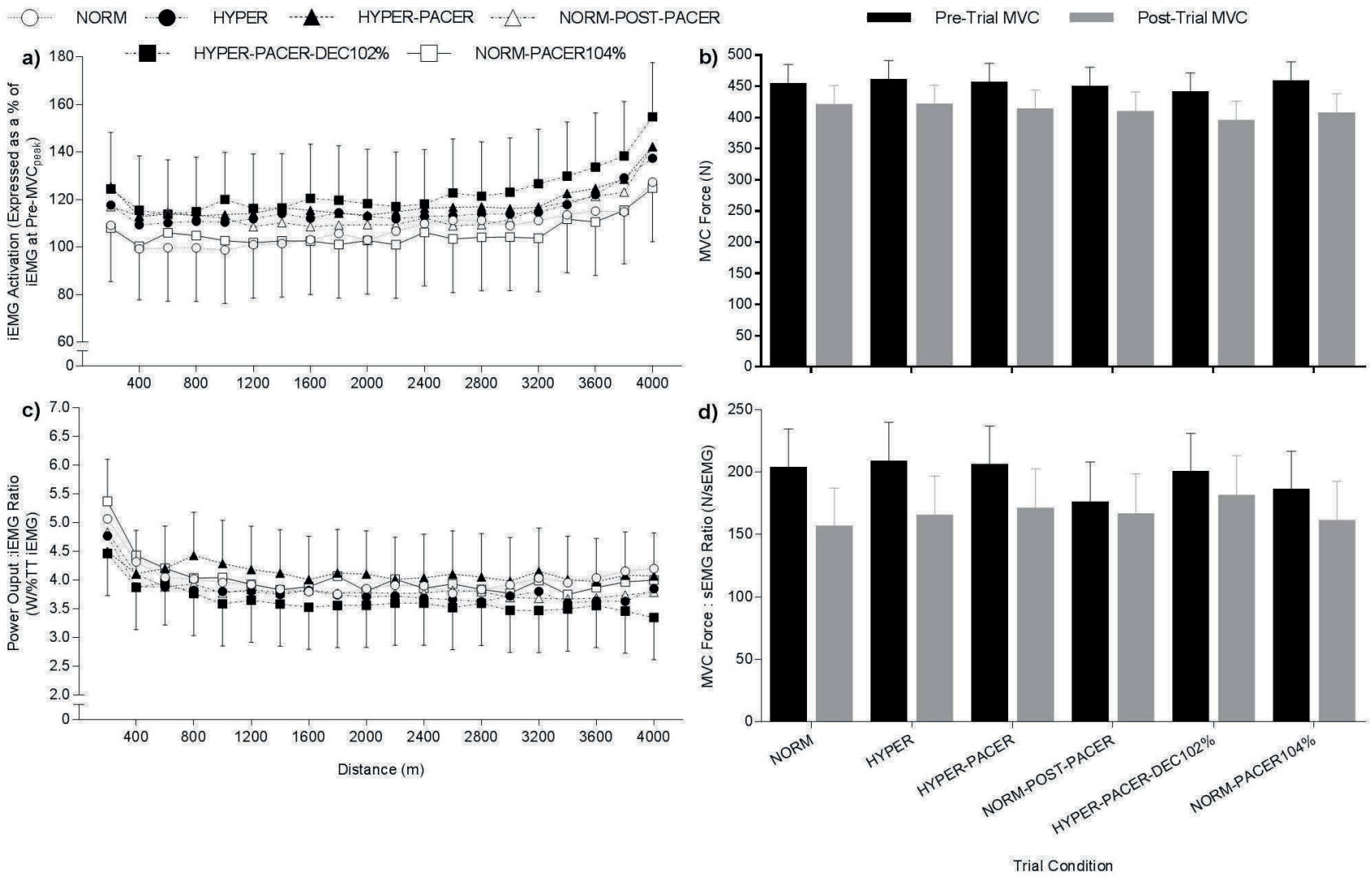


Figure 5.5 Integrated electromyogram (iEMG) activity in the right vastus lateralis leg muscle (**a**), ratio of power output to iEMG activity (**b**) at 200-m intervals for each condition, peak force (**c**) and ratio of peak force to surface electromyogram (**d**) during pre- and post-trial maximal voluntary contractions for each condition. Results are reported as mean estimates ± 95% confidence intervals.

5.4.5 Motivation, emotion and perceptual responses:

There was no significant Condition by Segment interaction for RPE ($F_{18,594} = 0.77$, $P = 0.74$). However, a significant Condition ($F_{6,594} = 18.82$, $P < 0.00$) and Segment ($F_{3,594} = 504.20$, $P < 0.00$) main effects were found for RPE (Figure 6a), where higher levels of perceived exertion were found in NORM-PACER104% compared to NORM, after the first 1000-m. There was no significant Trial by Motivation interaction for both success and intrinsic motivation ($F_{36,1028} = 0.25$, $P = 1.00$). In addition, no significant Trial by Motivation interaction was found in the SEQ questionnaire for: anxiety, dejection, excitement, anger and happiness ($F_{36,1028} = 0.25$, $P = 1.00$). However, cyclists did report higher success compared to intrinsic motivation scores (**Figure 5.6b**).

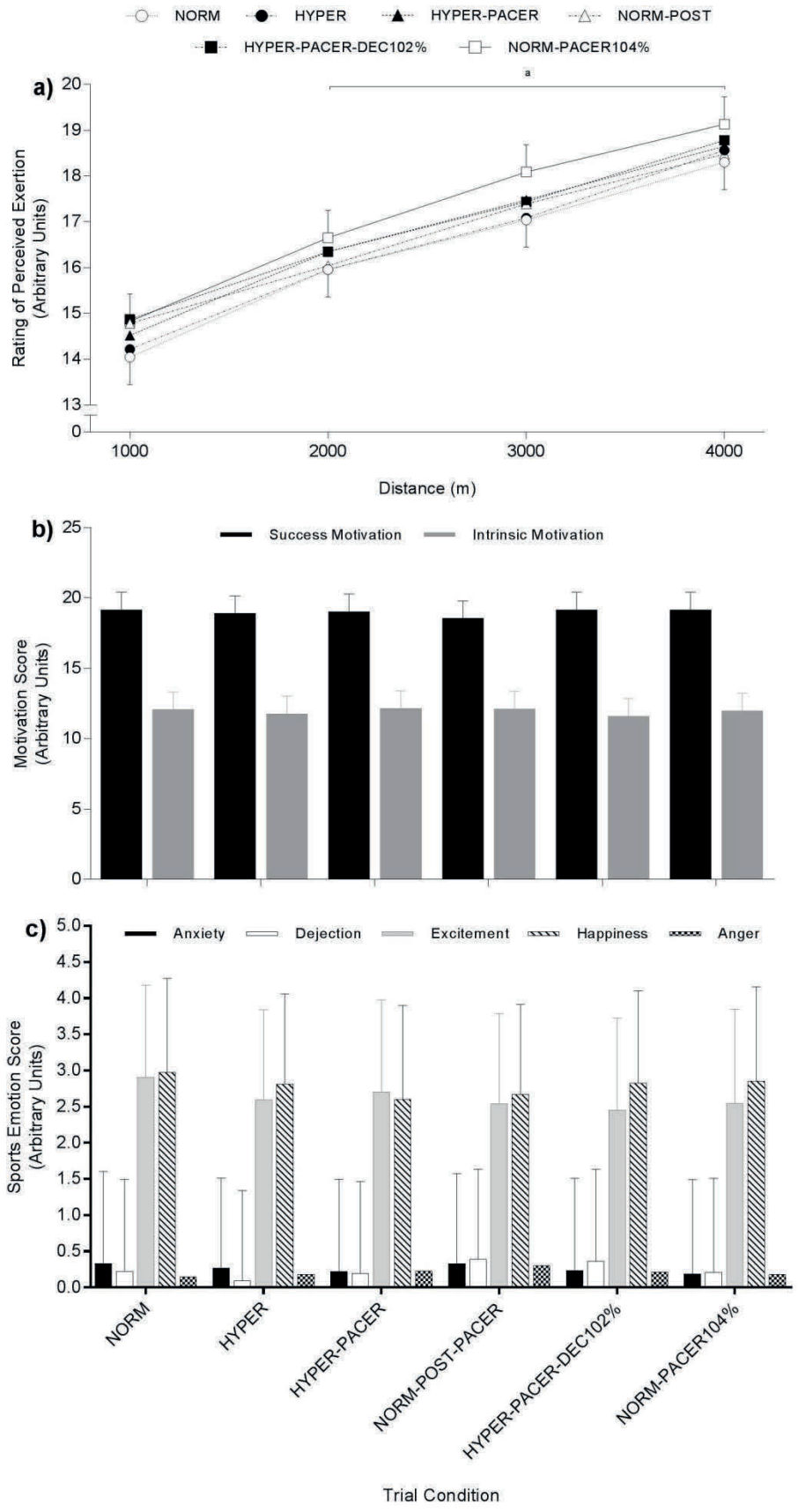


Figure 5.6 Ratings of perceived exertion at 1000-m intervals during 4000-m time-trials (**a**), pre-trial success and intrinsic motivation scores (**b**) and post-trial emotion scores (**c**) for each condition. Results are reported as mean estimates ± 95% confidence intervals. [b] Significant difference between NORM-PACER104% and NORM.

5.5 DISCUSSION:

Previously, negative feedback deception has been shown to elicit a significant performance improvement in normoxia (40, 89, 91, 158, 169, 203), however, to our knowledge, this is the first study to demonstrate negative feedback deception can improve performance in hyperoxia. Specifically, our results indicate cyclists were able to beat or match a pacer which deceptively misrepresented their previous best performance during a hyperoxic trial; improving TT performance by ~ 5 W or 2 s compared to a self-paced hyperoxic trial (HYPER) or by ~ 8 W or 3 s compared to a competitive hyperoxic trial where negative feedback was not given (HYPER-PACER). Performance in NORM-POST and NORM-PACER104% improved by ~ 7 W or 3 s and ~ 18 W or 7 s compared to NORM, respectively. Therefore, it appears prior performance improvements in hyperoxic trials might have influenced subsequent performances in normoxic TT's. Performance improvements in HYPER-PACER-DEC102% were associated with higher PO's during the first 800-m and greater P_{AER} to total PO compared to HYPER-PACER. Similarly, in NORM-PACER104%, a more aggressive starting strategy was observed compared to NORM and was associated with a significantly greater P_{AER} and P_{AN} contributions to total PO, an elevated cadence, $\dot{V}O_2$ and HR, as well as higher levels of perceived exertion. The changes in parameters in NORM-PACER104% improved performance compared to NORM, suggesting an exercise reserve was accessed in NORM-PACER104% which was likely influenced by the preceding HYPER-PACER-DEC102% trial.

5.5.1 Exploring the performance reserve in hyperoxia:

Based on previous studies, if cyclists were able to improve performance in HYPER-PACER-DEC102%, it would suggest they had accessed an exercise reserve when already in an ergogenic state from inspiring hyperoxic air (91, 158, 169). In the present study, it appears

that for most cyclists, the increased MPO of the pacer was tolerable, allowing an improvement in MPO by ~ 8 W above HYPER-PACER. Specifically, higher PO's were observed during the first 800-m in HYPER-PACER-DEC102% compared to a competitive hyperoxic trial with no deception. However, similar PO's were maintained thereafter for HYPER-PACER-DEC102% and HYPER-PACER. In addition, no significant differences in HR, S_pO_2 or RPE were found. Therefore, in support of previous work in normoxia (17, 89, 91, 158, 169, 171, 176, 177), these findings further suggest the subtle upregulation in exercise intensity from negative deception feedback is manageable to improve TT performance. A possible explanation for the observed improvement is the 2% increase in the pacer evoked a faster start from cyclists. Specifically, Konings et al. (95) demonstrated a faster starting pacer encourages greater MPO's by cyclists in the initial 750-m during 4000-m cycling TT's. However, Konings et al. (95) found similar performance times compared to a self-paced TT irrespective of the pacer behaviour. Critically, it appears deception of the pacer MPO in the current investigation was subtle enough that cyclists were unable to detect they had been deceived such that cyclists still initiated an end spurt in HYPER-PACER-DEC102%, which occurred slightly earlier compared to HYPER-PACER. An additional explanation is that finite capacities from the anaerobic energy system were accessed by cyclists at ~ 65–70% of the 4000-m distance, slightly earlier than ~ 80–90% suggested in normoxia (169). However, we were unable to detect a difference in the energy contributions between a known and deceived pacer when inspiring hyperoxic air; a finding contrary to earlier studies where normoxic performance deception evoked an increase in P_{AN} contributions (169). Consequently, the increased P_{AER} contribution to PO by ~ 9 W over the HYPER-PACER-DEC102% trial, suggests the 2% MPO increase to the pacer in HYPER-PACER-DEC102% was too low and we potentially could have increased the magnitude of deception above HYPER-PACER to further exploit anaerobic energy pathways.

5.5.2 Exercise regulation in normoxia after hyperoxic deception:

Interestingly, 19 of the 23 cyclists improved performance in NORM-PACER104% by ~ 18 W or 7 s compared to NORM. Similar to previous reports (158), the performance improvement was likely the result of a faster starting strategy in the first 1000-m, where a subtle upregulation in the pacing index and cadence were found from 400-m to 1000-m. Therefore, in agreement with earlier findings (158, 169), our results further suggest improved performance in a previous deception TT can be retained in a subsequent informed trial, where an increased belief or confidence may have been developed prior to NORM-PACER104%. Therefore, it is possible knowledge of previous trial performance, compared to perception of effort during the final trial, including knowledge of the MPO increase to the pacer, may have influenced cyclist's belief such that exercise intensity was upregulated (120, 199). Concomitant to performance changes was a significantly greater mean P_{AN} contribution in the first 2000-m, as well as significantly elevated $\dot{V}O_2$ and HR. In contrast, at the same time, S_pO_2 was significantly lower, where the higher intensity and HR possibly led to a greater degree of exercise induced hypoxemia contributing to a reduced S_pO_2 (10). It is therefore possible the faster starting strategy in NORM-PACER104% decreased the magnitude of the oxygen deficit incurred across the onset of exercise causing $\dot{V}O_{2peak}$ to be reached more rapidly as previously reported (27). As a result, despite similar P_{AN} energy contributions between NORM-PACER104% and NORM in the second half of 4000-m TT's, it is possible finite anaerobic energy pathways were made available and exhausted earlier in NORM-PACER104% which are often left in reserve during self-paced TT's (27, 166). Consequently, cyclists who made greater use of their anaerobic energy contribution in the first half of the final trial were unable to initiate an end-spurt ~ 800-m from the finish as performed in NORM. However, despite this occurrence, and contrary with earlier suggestions, overall performance was improved (27, 79, 169, 171). Therefore, the higher-than-usual exercise

intensity in the early stages of NORM-PACER104% may have built enough of a competitive advantage in the final stages of the TT, where cyclists believed they were capable of winning (95, 143). Such a decision to maintain the current work-rate was likely based on cyclists attempting to control the race relative to the position of the pacer (i.e. in front, matched or behind), motivation levels and proximity from the finish line (171). Moreover, it appears knowledge of the pacer MPO may have reduced the level of uncertainty in NORM-PACER104%, where despite cyclists reporting aligned RPE with intensity increases across the other trial conditions, in NORM-PACER104%, the higher levels of perceived exertion may suggest the increased work-rate was manageable (17, 146). This finding contrasts previous literature which suggested attention and the visualisation of a competitor provided a distractive mechanism, whereby exercise intensity increases in the absence of an increased RPE(199). Overall, the novel finding for the current investigation is a hyperoxic trial where cyclists received negative performance feedback appears to have improved performance in a competitive normoxic TT.

5.5.3 Effects of preceding hyperoxic time-trials on normoxic performance:

A secondary aim of the current study was to determine if inspiring hyperoxia in preceding trials may deviate cyclists from their routine pacing strategy and improve subsequent, self-paced normoxic performance. A significant improvement of ~ 7 W or 3 s was found in NORM-POST compared to NORM. Therefore, the order of trials preceding NORM-POST, may provide further evidence that environmental manipulation, with and without deception, has the potential to either improve belief or alter task expectations and improve performance. A similar finding has been previously shown in hypoxia, where cycling TT's at moderate, simulated altitudes disrupted the pacing template in subsequent trials at lower altitudes (34).

It is therefore possible previous hyperoxic performances may have influenced the cyclists belief such that exercise intensity was upregulated and TT performance improved (120).

5.5.4 Energy contributions in hyperoxia:

A novel aspect of the current investigation was the quantification of the P_{AER} and P_{AN} energy contributions in hyperoxia. Our findings suggest an increased P_{AER} contribution improved time to completion in HYPER, with a ~ 23 W increase in contribution to overall PO from the aerobic energy system compared to NORM. Consequently, inspiring supplemented oxygen significantly increased oxygen uptake and improved $\dot{V}O_2$ kinetics as described previously (137). This was likely caused by a greater cellular energy metabolism dependence on oxygen (105), reducing the reliance on P_{AN} energy contributions and biochemical and physiological disturbances to homeostasis (187). In comparison, a greater P_{AN} contribution was found in NORM-PACER104%, likely explaining the performance improvement similar to previous normoxic deception TT's (169). However, in the current investigation we found the P_{AN} contribution to be similar between HYPER-PACER and HYPER-PACER-DEC102%. It is possible the P_{AN} contribution may have been underestimated compared to earlier reports (43, 169), as although we measured GE in accordance with previous normoxic TT methodologies, the limitation of these methods is the assumption GE is the same during low-intensity steady state cycling compared to TT performance. Recent research has demonstrated GE decreases with fatigue and over time (43). Consequently, using a constant GE value may have led to underestimation of P_{AN} contributions. Therefore, despite changes in efficiency previously reported to not introduce meaningful errors into the calculation of P_{AN} contribution (66), findings should be interpreted with caution. Nevertheless, the current investigation does indicate differences in energetic contributions of energy systems between deception trials completed in hyperoxia compared to normoxia.

5.5.5 Voluntary force production and muscle activation:

In agreement with earlier research our findings suggest a greater muscle activation is associated with inspiring hyperoxic air compared to normoxia (187). However, like previous findings, changes in muscle activity across all trials were consistent, regardless of the Segment or the FiO_2. In addition, the magnitude of force reduction in post-trial MVCs was similar across all conditions, which suggests hyperoxia did not decrease the rate of development of peripheral muscle fatigue compared to normoxia (187). The central drive in motor unit recruitment or the frequency rate of motor units at the measured working musculature may have remained similar regardless of condition (70, 187). We acknowledge there is inherent measurement variability when sampling iEMG activity during dynamic exercise, often resulting in interpretation difficulties (170). Nevertheless, the observed changes in iEMG amplitude may still provide a practical reflection of changes in muscle activation during dynamic exercise (187).

5.5.6 Research limitations:

Previous studies have demonstrated that simulated head-to-head competition against a virtual on-screen pacer exerts a positive effect on performance (36, 89, 94, 95). In contrast, no difference in performance was found between HYPER and HYPER-PACER TT's despite accounting for the competitive influence of a second on-screen rider in hyperoxia. However, the current investigation did not include a condition in which cyclists performed a non-paced, non-competitive, normoxic time-trial. Consequently, the presence of an on-screen pacer may have provided additional motivation and may partly explain the increases in PO observed in the final trial (89, 91). Future research should therefore aim to further understand the implications of deception or belief on subsequent performances and whether improvements, or not are sustainable long term. As a result, whether improvements in a hyperoxic deception

trial can be sustained in a subsequent, self-paced normoxic TT remains unknown. Finally, as previously mentioned the anaerobic contribution to PO was potentially underestimated and should be interpreted with caution.

5.6 CONCLUSION:

For the first time, we show an exercise reserve may exist in hyperoxia, enabled by higher MPO's during the start of trial. In addition, a hyperoxic trial where cyclists received negative performance feedback appears to have improved performance in a competitive normoxic TT. The improvement in NORM-PACER104% was attributable to a more aggressive starting strategy concomitant to greater MPO, $\dot{V}O_2$, HR, RPE P_{AER} and P_{AN} contributions to total PO.

6.0 DISCUSSION OF THESIS

6.1 SUMMARY OF KEY FINDINGS

This body of work aimed to examine how environmental and performance feedback manipulations could affect exercise regulation and performance in trained cyclists. Three studies were undertaken to achieve this aim and will be summarised in the following sections. Additionally, a visual flow chart representing the thesis conception and progression is provided in **Figure 6.1** (page 164).

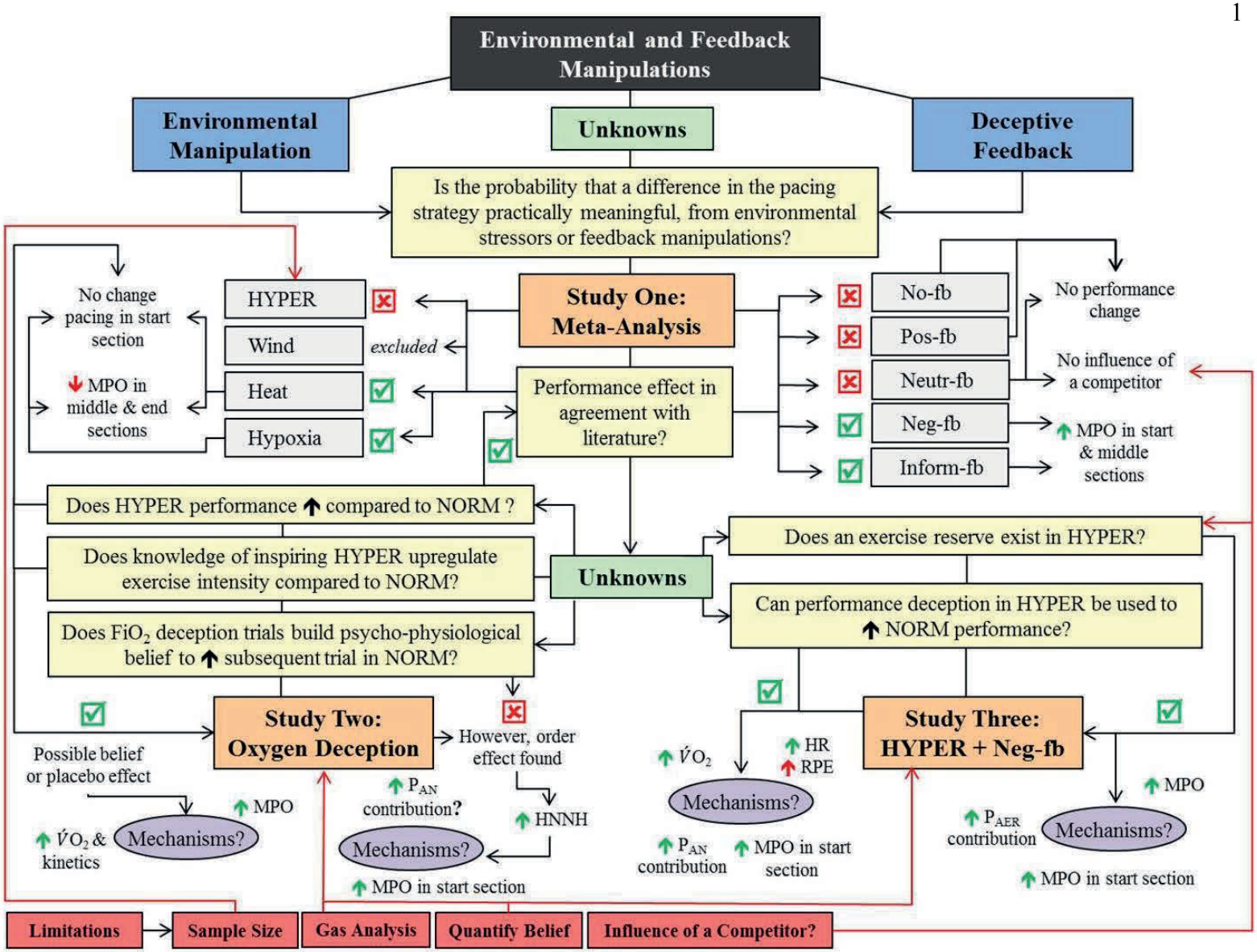


Figure 6.1 Visual flow chart on thesis conception and progression; ☑ represents a significant effect; ☒ represents no significant effect; ↑ indicates a significant improvement; FiO_2 = fraction of inspired oxygen; HR = heart rate; MPO = mean power output; P_{AER} = aerobic energy contribution; P_{AN} = anaerobic energy contribution; RPE = ratings of perceived exertion; $\dot{V}O_2$ = minute oxygen consumption; HYPER = hyperoxic trial; HNNH = oxygen inspired trials completed in order of: NORM-DEC, NORM, HYPER-DEC, HYPER; NORM = normoxic trial; 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.

6.1.1 Study One: summary of key findings

Surprisingly, despite a number of recent reviews attempting to summarise and explain pacing in self-paced exercise, the probability of whether a particular intervention is practically meaningful has been lacking, possibly due to the various forms in which exercise regulation is manipulated and examined. Therefore, the aim of Study One was to estimate the probability that a difference in pacing, due to environmental and feedback manipulations, is practically meaningful. To achieve this, a comprehensive meta-analysis was conducted. Overall, Study One found a negative effect of reduced oxygen availability and heat-stress on self-paced cycling TT's. In contrast, no segment or overall performance improvements were associated with cyclists inspiring supplementary oxygen, contrary to previous work reporting the ergogenic benefits of inspiring hyperoxia during TT's (10, 110, 126, 135-137, 187). However, this might have been attributable to the small sample size, warranting further research to determine the efficacy of inspiring hyperoxic air, how exercise is regulated and the implications to overall performance. Critically, the effects of environmental stressors (hypoxia and heat-stress) seem to only manifest in the second and third sections of performance trials compared with normoxic or cool conditions. These findings are in agreement with earlier reports, suggesting cyclists are preconditioned to select a similar starting strategy in different environmental conditions before afferent feedback from the periphery is assimilated and acted upon about whether the current exercise intensity should be maintained or adjusted (185).

A further finding from the meta-analysis was negative deceptive feedback may improve overall TT performance compared to other forms of feedback manipulation. Specifically, the provision of negative performance deception may increase MPO at the start and middle stages

of ‘deception’ trials. Improvements in performance are believed to be the result of athletes accessing an “exercise reserve”, likely the product of a greater anaerobic energy contribution to PO in later stages of a trial (169, 171). In contrast, the meta-analysis did not find an improvement for the end section of trials. However, this might be the result of segmenting TT’s into thirds, thus reducing the resolution of the analysed pacing profiles (169, 171). Nevertheless, similar to earlier reports, Study One did find practically meaningful changes in pacing and performance in a subsequent trial after cyclists were informed they had been deceived of the MPO increase to the pacer in a previous TT (158, 169).

6.1.2 Study Two: summary of key findings

Results from the meta-analysis indicated the provision of negative performance deception improves performance, both within the deception trial itself and during subsequent trials when deception is revealed. However, whether deception regarding environmental conditions could elicit similar responses to negative performance deception remained largely unknown. Previous studies blinding athletes to the FiO_2 indicate even slight increases to oxygen availability are sufficient to improve performance by 3–14% compared with normoxic trials (10, 105, 137, 187). However, to our knowledge, no current research has described the effects of FiO_2 deception on athletic performance. Therefore, whether knowledge of inspiring hyperoxia encouraged an upregulation of exercise intensity compared to a normoxic trial required further exploration. To address this, Study Two aimed to determine whether a series of trials manipulating FiO_2 and knowledge of the inspired oxygen content (through deception of the inspired air) might alter the pacing profile and performance during 4000-m cycling TT’s. In general, we found that cycling performance improved when cyclists inspired hyperoxic air, irrespective of whether they were aware of the inspired oxygen or not. Furthermore, when participants were asked whether they were aware or suspected they had

been deceived of the inspired oxygen content they were unable to correctly identify inspired gas content. Mechanisms for the improvement were likely and predominantly physiological. For example, increasing oxygen availability has been attributed to an increased $\dot{V}O_2$ and faster $\dot{V}O_2$ kinetics (137), increased oxygen availability at skeletal muscles and reduced biochemical and physiological disturbances to homeostasis (187). However, the subtle difference in the magnitude of MPO improvement in an accurate, compared to a deceptive hyperoxic trial suggests an additional placebo effect may also be present. In contrast, a series of oxygen deception trials did not improve performance in a subsequent, normoxic TT once deception was revealed. However, cyclists who completed trials in the following order NORM-DEC, NORM, HYPER-DEC, HYPER (HNNH) appeared to build psycho-physiological belief and improved performance in a subsequent normoxic trial (NORM-INFORM). Performance improvements for this specific group were attributable to a more aggressive starting strategy and coincided with an elevated HR in NORM-INFORM.

An additional aim in Study Two was to examine whether environmental deception changed pacing during the deception trials themselves. In a normoxic-deception trial, cyclists maintained greater MPO's compared to NORM across all orders. Interestingly, in agreement with results from Study One, while MPO did increase in the normoxic-deception trial, initial PO was similar to a known normoxic trial. Therefore, these findings support earlier conclusions that athletes are accustomed to start self-paced TT's at a similar work-rate despite manipulations to FiO_2 (78, 87, 137, 187) Nevertheless, as the trial continued, cyclists belief they were inspiring normoxia did not blunt the benefits of hyperoxia, a finding contrary to many models and theories of exercise regulation which emphasise the importance of prior experience overcoming peripheral feedback (117).

Of interest, cyclists who completed order HNNH also improved MPO in a hyperoxic-deception trial compared to NORM. As a result, cyclists in this trial were able to mitigate the limitations of inspiring normoxia when believing they were inspiring hyperoxia, suggesting this belief influenced their performance. Consequently, the order in which trials were completed in HNNH may have been sufficient to disturb cyclist's routine pacing template, causing PO to be upregulated in a non-ergogenic normoxic environment. This explanation is further supported by an upregulation in MPO between 1600-m to 4000-m for all TT's in order HNNH compared to other orders, suggesting the FiO_2 in preceding TT's may have altered pacing in subsequent trials. Moreover, the order of trials preceding the improvement in NORM-POST in Study Three, provides further evidence that environmental manipulation, with and without deception, has the potential to either improve belief or alter task expectations and improve performance – a finding previously shown in hypoxia, where cycling TT's at moderate, simulated altitudes disrupted the pacing template in subsequent trials at lower altitudes (34). It is therefore possible trial orders implemented across the two different studies may have influenced the cyclist's belief such that exercise intensity was upregulated and TT performance improved (120).

6.1.3 Study Three: summary of key findings

In contrast to the meta-analysis, Study Two confirmed earlier opinions that supplemental oxygen permits a greater exercise capacity (110). Furthermore, Study One affirmed negative deceptive feedback is sufficient to upregulate MPO and elicit a significant performance improvement in normoxia but not so great as to be intolerable (40, 89, 91, 158, 169, 203). Earlier research in normoxic TT's endorse the notion that finite anaerobic energy supplies can be accessed when athletes receive negative performance deception; often left in reserve during self-paced TT's (169, 171). However, whether an exercise reserve exists in hyperoxia

remained largely unknown. To address this, the primary aims of Study Three was to investigate i) if a performance reserve exists in hyperoxia and ii) whether negative performance feedback could be used in hyperoxia to change task expectations and build psycho-physiological belief such that performance in a subsequent normoxic TT was improved.

Performance deception in hyperoxia was achieved in the form of an on-screen avatar (pacer) whose PO was set 2% above a previous competitive hyperoxic trial, similar to previous investigations in normoxia (40, 89, 91, 158, 169, 203). To our knowledge, this is the first study to demonstrate negative performance deception can improve performance when inspiring hyperoxic air. During this trial (HYPER-PACER-DEC102%), for most cyclists, the increased MPO of the pacer was tolerable, allowing an improvement in MPO by ~ 8 W above a competitive hyperoxic trial with no deception (HYPER-PACER). Notably, higher PO's were observed during the initial segments of HYPER-PACER-DEC102% compared HYPER-PACER, supporting the meta-analysis, where negative performance deception allows a subtle upregulation in MPO during the initial stages of 'deception' trials. However, contrary to previous reports and findings from Study One, similar PO's were maintained thereafter for HYPER-PACER-DEC102% compared to HYPER-PACER (89, 91, 169, 171, 176, 177, 203).

In support of previous negative deceptive performance trials in normoxia (17, 89, 91, 158, 169, 171, 176, 177), no significant differences in HR, S_pO_2 or RPE were found between HYPER-PACER-DEC102% and HYPER-PACER, suggesting the subtle upregulation in HYPER-PACER-DEC102% was manageable to improve TT performance. In addition, participants reported being unaware they had been deceived of the manipulated pacer. As a result, cyclists still initiated an end spurt in HYPER-PACER-DEC102% which occurred

slightly earlier compared to HYPER-PACER. Furthermore, the end spurt found in the competitive, hyperoxic deception trial was initiated earlier than would be expected had the trial been in normoxia (169). An explanation is that finite capacities from the anaerobic energy system were accessed by cyclists at ~ 65–70% of the 4000-m distance, slightly earlier than ~ 80–90% suggested in normoxia (169). However, we were unable to detect a difference in anaerobic energy contributions despite earlier studies suggesting normoxic, negative performance deception evokes an increase in P_{AN} contributions (169). Instead, there was an increased P_{AER} contribution to PO by ~ 9 W over the HYPER-PACER-DEC102% trial, suggesting improved oxygen availability may have mitigated any increase in anaerobic energy contribution. Therefore, it is possible the 2% MPO increase to the pacer in HYPER-PACER-DEC102% was too low and we potentially could have increased the magnitude of deception to further exploit the exercise reserve.

In agreement with earlier investigations (158, 169), Study Three established performance improvements in a previous hyperoxic deception TT can be retained in a subsequent informed trial. Performance improvements were associated with a faster starting strategy in the first 1000-m, where a subtle upregulation in the pacing index and cadence were found from 400-m to 1000-m. Therefore, similar to improvements in the NORM-INFORM trial for a particular order of trials found in Study Two, knowledge of previous trial performances, matched with moment-to-moment perception of effort during the final trial in Study Three, may have influenced cyclist's belief such that exercise intensity was upregulated (120).

Concomitant to performance changes in NORM-PACER104%, was a significantly greater mean P_{AN} contribution in the first 2000-m, as well as significantly elevated $\dot{V}O_2$ and HR. As a result, oxygen uptake and $\dot{V}O_2$ kinetics significantly increased following the onset of exercise,

where it is possible the exercise reserve was exhausted earlier in NORM-PACER104% compared to NORM despite similar P_{AN} contributions in the second half of 4000-m TT's (27, 166). Consequently, cyclists who made greater use of their energy reserve in the first half of the final trial were unable to initiate an end-spurt ~ 800-m from the finish as they could in NORM. However, despite this occurrence, and contrary with earlier suggestions, overall performance was improved (27, 79, 169, 171). The higher-than-expected exercise intensity in the early stages of NORM-PACER104% may have built enough of a competitive advantage in the final stages of the TT, where cyclists believed they were capable of winning (95, 143). Such a decision surrounding cyclists ability to maintain the current work-rate was likely based on their attempt to control the race relative to the position of the pacer (i.e. in front, matched or behind), motivation levels and proximity from the finish line (171). Therefore, reducing cyclists uncertainty surrounding whether the increased work-rate was manageable despite cyclists reporting higher levels of perceived exertion (17, 146). Overall, the novel finding for Study Three is a hyperoxic trial, where cyclists received negative performance feedback, appears to have improved performance in a competitive normoxic TT.

6.1.4 Theories and models of pacing and exercise regulation

This body of work has provided valuable insight into the mechanisms which affect how athletes pace during exercise and provides further evidence to both support and refute different models of pacing regulation. Collectively, and in agreement with the perception based model (183), the current thesis has demonstrated that cyclists are preconditioned to select a similar starting strategy in different environmental conditions before afferent feedback from the periphery is assimilated and acted upon to adjust exercise intensity (185). Moreover, the current thesis supports the existence of a pre-determined pacing template described by Ulmer's (190) model which suggested athletes anticipate the work required to

complete an exercise task before work output is distributed accordingly. Furthermore, findings from Study Two revealed that cyclists were likely more conservative after a negative pacing experience in a previous trial, highlighting the importance of prior knowledge and experience on modifying athletes pacing template which may not be completely fixed as once thought (130). Contrary to this theory was the influence of afferent feedback in the NORM-DEC trial in Study Two. During this trial participants were informed and believed they were inspiring normoxia, when in fact hyperoxia was inspired, suggesting afferent feedback was sufficient to overcome prior knowledge and belief such that PO was still increased in the NORM-DEC trial (70, 129, 130). Deceiving cyclists of their MPO achieved in a previous performance, Study Three, further demonstrated that perception of effort is an important mediator in detecting mismatches with pre-exercise expectations surrounding the current performance, before selection of an appropriate pace is chosen based on incoming internal and external stimuli (190). This phenomena has been proposed as not simply a downregulation from central to peripheral centres, previously proposed by Noakes and colleagues (130), instead it is an ongoing and complex interaction between the CNS (i.e. brain) and incoming peripheral feedback (165). Subsequently, athletes make decisions about whether the current exercise intensity should be maintained or adjusted in a decision-making process which may be spontaneous or deliberate, under periods of certainty or uncertainty, in an attempt to evaluate the level of risk if the decision is acted on (69). For instance, in Study Three cyclists still initiated an end spurt in HYPER-PACER-DEC102% which occurred slightly earlier compared to HYPER-PACER. However, despite current models primarily agreeing pace selection is determined by an athlete's ability to resist fatigue, the precise mechanisms of exercise regulation remain unclear. Furthermore, current models and theories appear to contradict each other. For example, although it seems apparent athletes pacing template is pre-determined in the initial stages of a performance bout, afferent feedback can

still influence selection of pace, as shown in Study Two, if incoming internal and external stimuli creates a mismatch with the current performance. Therefore, further research is still needed to understand the complexity of exercise regulation, particularly surrounding central and peripheral fatigue and decision-making processes as they occur during a variety of exercise modalities, conditions and durations.

6.2 PRACTICAL APPLICATIONS

Given the implications of pacing to overall performance, from a practical perspective, Study One demonstrated the importance for coaches and athletes to consider adapting the 'normal' race pacing strategy to account for environmental stressors. For instance, adapting the routine pre-determined template to the current thermal stress may minimise an error in pace judgment occurring from the start of the race. Otherwise an excessive heat-stress may occur, severely reducing exercise intensity and negatively impacting performance. Athletes race preparation in heat should therefore include practicing different pacing strategies in the competition environment (in location or simulated) to ensure an appropriate pacing strategy is in place.

Similarly, coaches and athletes need to consider an appropriate pacing strategy when training or competing at altitude, to avoid misjudging pace selection from the start of the race which may have negative consequences on overall performance. For athletes who compete globally, consideration should be taken of the effects training and competitions at altitude have on lower altitudes. For instance, if the experience is negative, future sessions or performance may be impacted by a conservative pacing strategy as previously reported (34). Therefore, thought into how best to maximise periods of training and competitions at differing altitudes is advised. Current research demonstrates a linear ~ 7% reduction in TT performance per 1000-m of altitude gained during self-paced exercise in hypoxia (45). Therefore, a 3000-m increase in altitude from near sea level (~200-m), such as during the Qinghai Lake International Cycling Race (altitude of 3600- to 5000-m meters), can expect a ~ 21–35% decrease in overall performance. A logical progression to overcome the negative effects of hypoxia in training or competition, other than acclimatisation, might be a gradual exposure to higher levels of altitude. For instance, Clark et al. (34) highlighted 1000-m increases at simulated altitude from 200-m caused minimal disturbance to the pacing template during a

five minute cycling TT compared to randomised levels of altitude at 200-m, 1200-m, 2200-m and 3200-m in elite cyclists. As a result, a suggested recommendation is to progressively increase hypoxic levels from near sea level giving athletes the best possible opportunity to maximise training, competition events or both.

Practical findings from Study Two were consolidated further where it appears hyperoxic trials and oxygen deception may be used as a potential method to alter task expectations, belief and cause disruption to the routine (pre-determined) pacing template to improve subsequent normoxic performance or competitive events. Therefore, the order in which hyperoxic and deception TT's are performed may be used to encourage athletes to deviate from their routine pacing template.

Finally, it seems apparent the provision of deceptive information, specifically negative performance deception is the most effective method of feedback manipulation to change pacing and performance. Similarly, if athletes are exposed to negative performance deception in hyperoxia, energy reserves that may be accessed often left in reserve during self-paced exercise. In comparison, an exercise reserve may also be accessed when cyclists complete oxygen deception trials in a particular order. Further discussion regarding opportunities to improve self-paced exercise performance and suggestions for use of hyperoxia and deception to access an exercise reserve will be discussed in Section 6.5.

6.3 STUDY LIMITATIONS

The studies presented in this thesis were conducted using pre-existing data from the literature or examining exercise regulation in a controlled laboratory setting with trained cyclists. Despite endeavouring to apply a high degree of scientific rigour to the studies, the chosen research designs and methodologies resulted in several limitations.

A limitation of the meta-analysis (Study One) was by only examining pacing in cycling, generalisation pertaining to exercise regulation to other modes of exercise such as running, swimming, skating and rowing is limited. As a consequence of these constraints, studies suitable for a meta-analysis in other modalities are worth further investigating, as they may provide further understanding of how athletes regulate exercise within the pacing literature. For instance, earlier work has demonstrated certain locomotive events, such as swimming (183), relay athletics (150) and rowing (65), involve specific starting procedures which allow maximal velocity or encourage speeds to be achieved in a shorter time. Therefore, analysis for the modalities described above may provide additional insight into exercise regulation and performance. Moreover, the current analysis only included simulated environmental conditions in a controlled laboratory setting. Consequently, whether similar pacing patterns occur in a variable outdoor environment can only be generalised at this point in time. Nevertheless, findings related to exercise regulation from the meta-analysis provide further insight into earlier work supporting the negative effects of hypoxia and heat-stress on pacing and performance (10, 45, 134, 189). Similarly, recent meta-analyses and reviews endorse the benefits of hyperoxia and negative performance deception during cycling TT performance demonstrated in Study One, respectively (90, 110, 204).

A further limitation was studies were discounted if they did not report PO 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, several studies providing cycling velocity or performance time data were excluded, despite meeting all other criteria. Additionally, the pacing resolution in the meta-analysis was compromised because trial PO data could only be segmented in to three sections (start, middle and end segments) 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 or distance and reporting conventions used in the included studies. Finally, with the exception of one study that recruited one female participant into a male cohort (139), only male participants were recruited in the included studies. Therefore, the findings have an obvious sex bias, as unfortunately there is a dearth of studies to date investigating changes in the pacing strategies exhibited by females.

The inclusion of respiratory gas data in Study Two would have provided additional insight into how cyclists pace when aware or not of the inspired oxygen content. Although we did attempt this across numerous pilot trials, technical limitations at the time with the chosen custom-built metabolic cart did not permit valid and reliable measurement of expired gas during the hyperoxic TT's. An additional limitation was the sample size (n = 15) in Study Two, where a maximum of four participants could be assigned to each group to account for a trial order effect when implementing the double-blinded and randomised order study design. Finally, a measure of cyclist's belief or competency was not quantified before the final trial. Therefore, we can only speculate the improvements in NORM-INFORM by those cyclists completing trial order HNNH was the result of belief built from proceeding trials.

Study Three was limited by the following conditions. Firstly, although the competitive influence of a second on-screen rider in hyperoxia was accounted for, the current investigation did not include a condition in which cyclists performed without a competitor in NORM-PACER104%. For example, a TT with no pacer could be used to determine with greater clarity whether belief was built from a previous negative deceptive performance trial in hyperoxia. This could be achieved by adding one additional trial without inspiring hyperoxic air or negative deceptive feedback manipulation. Consequently, the presence of an on-screen pacer may have provided additional motivation and may partly explain the increases in PO observed in the final trial (89, 91). As a result, whether improvements in a hyperoxic deception trial can be sustained in a subsequent, self-paced normoxic TT remains unknown. However, the meta-analysis supported earlier reports where the presence of an avatar did not influence the magnitude of the performance improvement compared to a control TT where no pacer was present (22, 157). Nevertheless, the influence of a competitor to date has been equivocal, warranting further investigation to advance our understanding of how competition is perceived.

Finally, although we measured GE in accordance with previous normoxic TT methodologies, the limitation of these methods is the assumption GE is the same during low-intensity steady state cycling compared to TT performance. Consequently, by using a constant GE value likely overestimated GE during hyperoxic trials in Study Three, particularly during the latter stages, and as a result P_{AN} contributions were likely to be underestimated and should be interpreted with caution.

6.4 FUTURE RESEARCH RECOMMENDATIONS

6.4.1 Future recommendations when investigating pacing

The findings presented within this thesis also point to several directions for future research. For example, 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 raw performance metrics (e.g. power output). The pacing index reports the exercise intensity for each segment as a percentage of overall trial MPO and is derived using the following equation:

*(Segment mean power output / Overall trial mean power output)*100*

The pacing index therefore allows for direct comparisons between sexes, age or in the instance of a meta-analysis, between different studies of similar themes. A further consideration is the calculation of a pacing index change score, as provided in Study One (**Figure 3.12**), which allows additional comparisons between trial conditions of where and when athletes distribute their energy. A graphical representation in **Figures 6.2** and **6.3** demonstrates the differences in pacing that are highlighted by the pacing index but not detected by visually examining PO alone. For example, on exploratory graphical inspection of the MPO distribution in the NORM trial (**Figure 6.2**), MPO is downregulated compared to other trial conditions (**Figure 6.2a**). In comparison, when accounting for the variability in the pacing strategy between trials, the pacing index demonstrates quite similar parabolic pacing profiles between conditions (**Figure 6.2b**). In addition, the notable changes in the starting strategy and blunted end spurt for NORM-INFORM becomes more evident, suggesting cyclists in NORM-INFORM employed a different pacing strategy in contrast to other trial conditions. Similarly, a graphical representation in **Figure 6.3** further demonstrates that

reporting the pacing index change score in heat highlights the pronounced reduction in MPO from thermal stress (32–35°C) compared to temperate (15–23 °C) conditions. Providing additional information of how athletes regulate exercise during the 40-km cycling TT's instead of reporting PO alone.

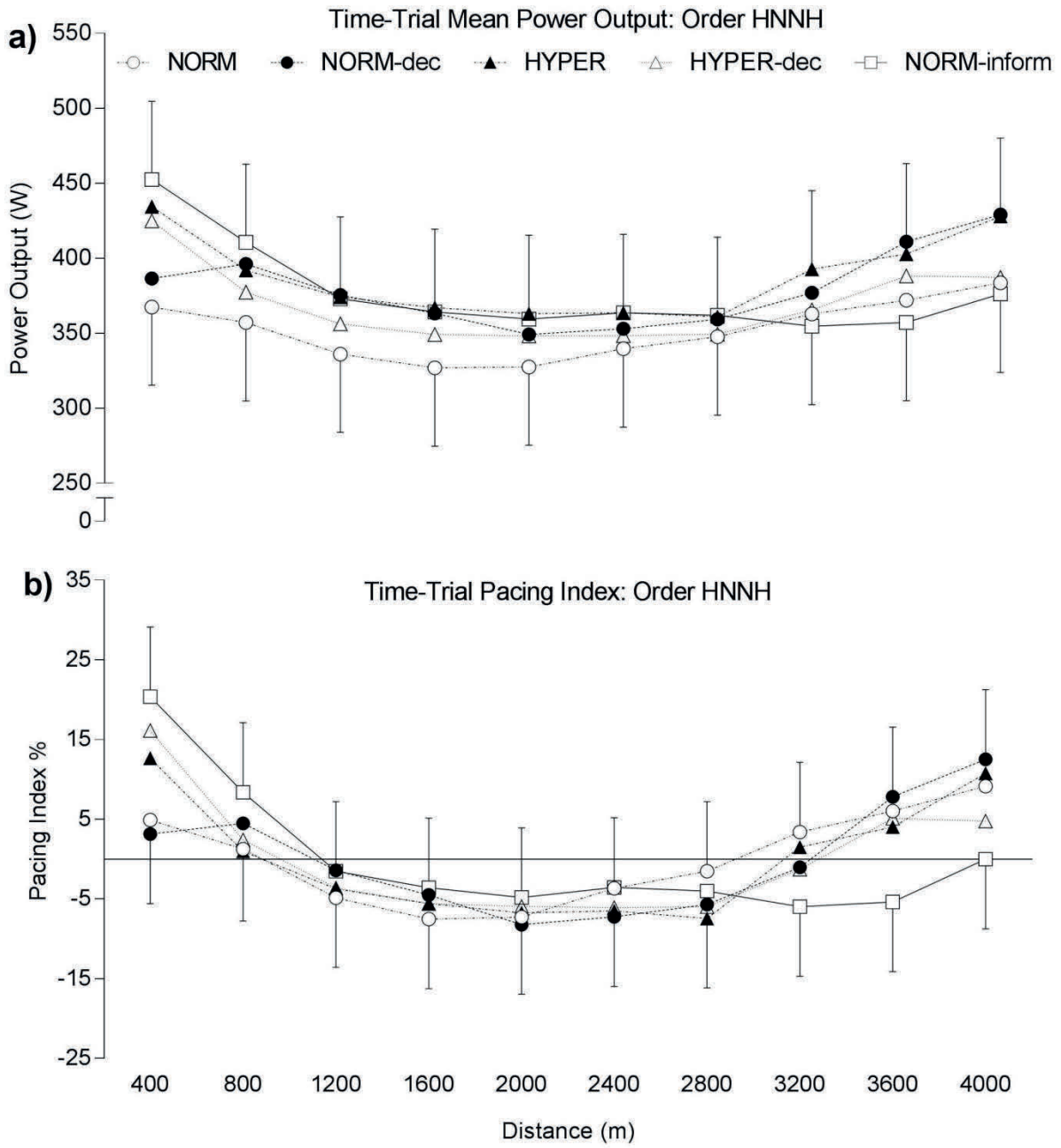


Figure 6.2 Graphical representation of how mean power output alone (**a**) cannot fully describe the variability in the pacing strategy during oxygen deception trials compared to reporting the pacing index (**b**) across 4000-m cycling time-trials.

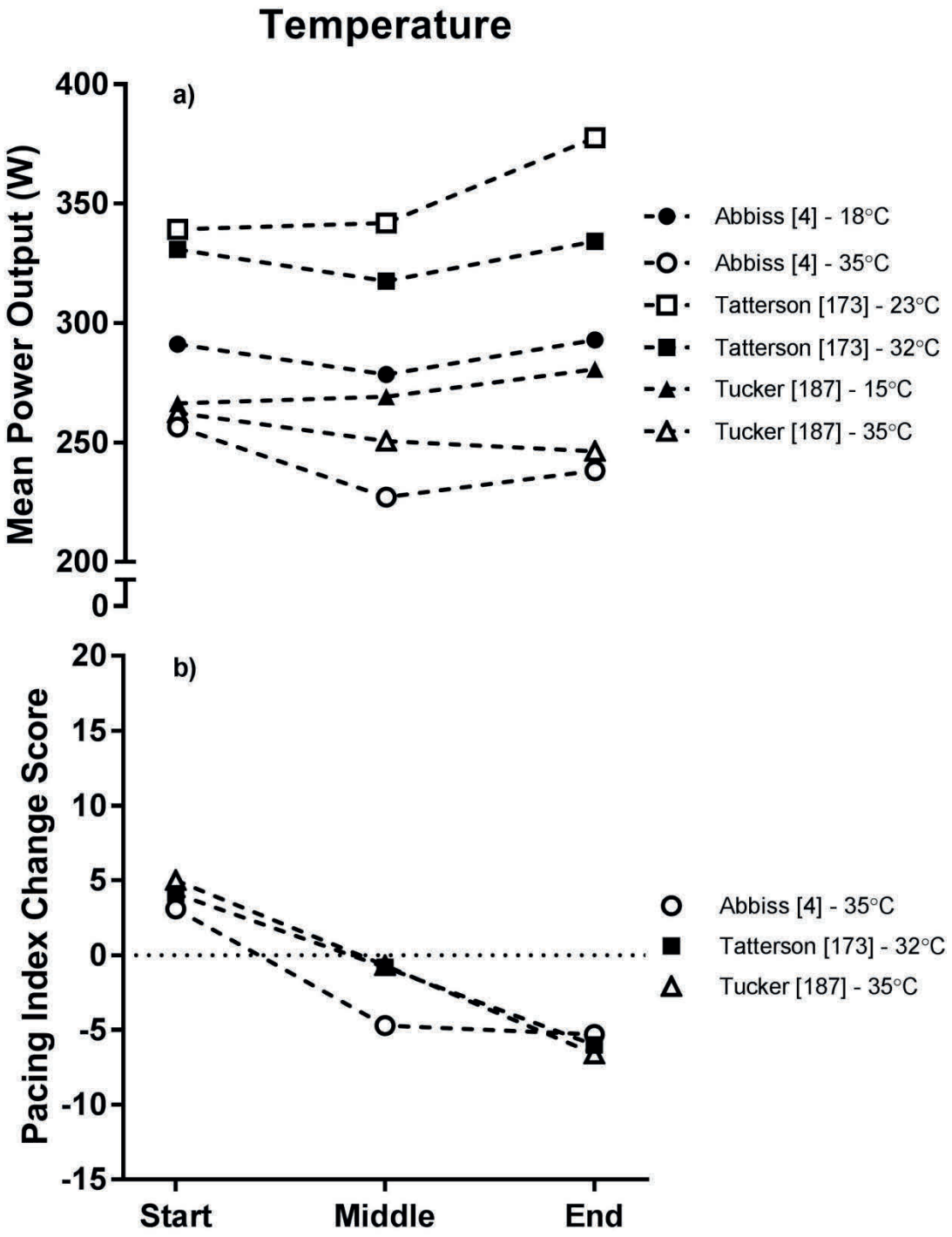


Figure 6.3 Graphical representation of how mean power output alone (**a**) cannot fully describe the variability in the pacing strategy in heat compared to reporting the pacing index (**b**) during the start, middle and end section of self-paced cycling time-trials. The presented data is from the meta-analysis (Study One).

6.4.2 Future recommendations for self-paced exercise when manipulating environmental conditions

Study One established the importance of considering the exposure time to heat-stress prior to and during a TT or a competitive race. Additionally, a number of specific considerations for experimental investigators were highlighted when undertaking heat-stress trials in laboratory settings: i) the amount of change in temperature between control and heat intervention trials; ii) whether the control trial represents conditions appropriate for a thermo-neutral trial; iii) the availability of fluids for *ab libitum* drinking; iv) the clothing worn; and v) adopting convective cooling methods to reflect equivalent volumes and rates of air flow that athletes encounter during outdoor competitions (~ 25–40 $km \cdot h^{-1}$ when cycling) (50).

Research studies manipulating FiO_2 should consider the potential effect trial orders may have on changing participants task expectations, knowledge or belief, and whether the study aims to disrupt pacing templates and overall performance during and in subsequent trials. Examples include, when simulated moderate altitude trials are followed or randomised with lower altitude trials or when randomising hyperoxic and hypoxic trials. More specifically, previous work performing TT's either at 3200- or 2200-m simulated altitudes, demonstrated a conservative approach in the start section of subsequent trials at lower simulated altitudes (200- and 1200-m respectively) (34). Similarly, Study Three demonstrated preceding TT's in hyperoxia altered performance in a subsequent normoxic trial, thus randomising hyperoxic and hypoxic TT's may have a more pronounced effect. In addition, future investigations should explore whether changes in pace and performance from a series of oxygen deception trials can be sustained over longer periods of time (e.g. throughout weeks to months of a competition season). Furthermore, investigations into the magnitude of deception in hyperoxia warrants further research to determine if anaerobic energy pathways can be

exploited which may provide additional insight into the physiological source of the energy reserve.

A further consideration for future studies manipulating oxygen availability is the potential effects of blinding participants, of the FiO_2 which may result in further alterations to pacing and performance. Moreover, exploration is required to understand the effects of high-altitude competition on pacing and performance on subsequent competitions at lower altitudes. However, little is known in this area and further insights may provide information to athletes and coaches on how to appropriately schedule competition events or training over the calendar year to potentially manipulate the pacing template or avoid suboptimal performances.

Finally, despite the current thesis providing insight into exercise regulation in hyperoxia, greater understanding is still needed to determine the effects that fatiguing exercise in hyperoxia has on GE, as it may assist with potential indicators for training. Practically, this will provide valuable information for coaches and athletes as to whether inspiring oxygen enriched air translates to a greater metabolic stimulus and ensure training intensities are matched correctly to the artificially enhanced environment. Otherwise there is a chance of athletes becoming overtrained more easily (i.e. increase muscle damage or greater neuromuscular strain to generating higher workloads), if care towards prescribing appropriate training intensities and recovery are not implemented (110).

6.4.3 Future recommendations for manipulating feedback in self-paced exercise

It seems apparent the provision of deceptive information, specifically negative performance deception is the most effective method of feedback manipulation to change pacing and performance. Future research should therefore aim to discover ways to ensure changes in pace and performance are sustained in subsequent trials and if changes can be sustained over longer periods of time (e.g. throughout weeks to months of a competition season) following negative performance deception. Additionally, the pacing literature could benefit from research investigating ways by which feedback could be used during competition to change performance. To achieve this, researchers could misinform athletes of physiological disturbances due to environmental stressors or manipulating performance metrics during competitive events. Examples include: deceiving athletes ambient or core body temperatures are lower than reality or manipulating power meters to under read PO by 2–4%, in an attempt to challenge athlete's task expectations, belief or goals to access reserve capacities and improve performance. However, despite improved cycling performance from deceiving cyclists of ambient temperatures compared to actual values (31), ambient deception increases the variability in PO which may become more problematic in regulating core body temperatures in heat over long duration events (> 30 minutes). Additionally, deceiving athletes of performance metrics in competition has the potential to cause: premature fatigue, resulting in suboptimal performances; an increased likelihood of muscle damage or soreness, posing challenges over multi-stage events; or damage working relationships between the coach, athlete and scientist. Consequently, whether feedback manipulations are a viable tactic to employ in competitions warrants care and further investigation.

6.4.4 Health and ethical considerations when administering supplemented oxygen

Increasing the FiO_2 above normal atmospheric levels imposes a unique demand on the cardiorespiratory system, where the duration and dosage level may have severe health implications (108, 110, 163). If respiratory muscles cannot manage the greater oxygen supply, cell damage or dysfunction may occur due to an elevated number of oxygen free radical bodies (108). More specifically, the alveolar epithelial and alveolar capillary endothelial cells, involved in gaseous exchange of oxygen into the blood capillaries, become vulnerable targets for oxygen free radical induced injury (108). To avoid such occurrences, medical recommendations for use of hyperoxia suggests gas contents do not exceed a FiO_2 of 0.60 when administered > 24-hours to avoid oxygen toxicity in the lungs leading to complications for the participant (108). The FiO_2 administered in the exercise science literature ranges (FiO_2: 0.24–1.0), during exercise bouts, recovery periods or both. Though some of the higher doses of hyperoxic air sit well above medical recommendations, the time period athletes have been exposed to hyperoxic air during exercise is significantly less compared to medical purposes (110). However, since the effects of chronic use of hyperoxic exposure on athlete's health remains unknown (i.e. as a training modality), it has been suggested the administration of hyperoxia be used in a controlled, regulated manner, preferably using lower oxygen contents, to avoid any potential harm (163).

Not surprisingly, the current use of supplemented oxygen is prohibited for use during competition. However, it still remains a legal practice as part of daily training environment for athletes (208). Although, Sperlich et al. (163) discusses not all athletes may have access to hyperoxic training methods and whether or not this creates an unfair playing field is open to interpretation. Nevertheless, The World Anti-Doping Agency's (WADA) guidelines are

constantly reviewed and updated with the changing athletic environment. Therefore, we recommend researchers, scientists, coaches, and athletes remain informed with local and foreign countries rules and regulations regarding the use of hyperoxia in training, including WADA's prohibited list to ensure no breaches of codes are made (208).

6.4.5 Ethical considerations when deceiving athletes

A concern when manipulating feedback in athletic research is whether methods conform to ethical guidelines. These manipulations include: withholding information, such as performance metrics (i.e. time, PO, velocity and HR), physiological responses, exercise duration or endpoint, or deliberately misinforming participants of the true purpose and procedure of the study. Consequently, participants are not provided complete information in the informed consent process. Therefore, based on previous guidelines and recommendations, it is critical researchers wishing to implement deception techniques consider: i) are there no reasonable alternatives for obtaining the data; ii) will participants dignity and autonomy be respected; and iii) if implemented can the risk of psychological harm or distress be minimised (1, 14, 76).

To determine the overall benefits of inspiring hyperoxic air and internal reserve capacities in the current investigation the deception techniques used were deemed safe and appropriate by a human ethics committee. This was achieved by providing false expectations towards environmental conditions and previous performances. As a result, research aims were protected from being revealed to participants. Importantly, no harm or distress was caused to participants by deception. Moreover, in accordance with ethical recommendations concerning deception (1, 14, 76, 90), participants were accurately informed of the risks and fully debriefed of the research aims at the conclusion of both intervention studies, with the option

to withdraw their data. Deception is considered inappropriate if any adverse effects on participation occur when revealing deception to participants (i.e. discomfort, anger, or objection) (1). If this was to occur, researchers have been recommended to provide a detailed account of debriefs to assist the design of future deception studies (90). Anecdotal evidence from debriefs in the current thesis suggested participants were unware of the deception, and in fact, participants were surprised to learn of the oxygen and performance deceptions in Study Two and Three, respectively. However, if deceptive methods are used appropriately by correctly debriefing athletes to the overall purpose of deception (i.e. to exploit their energy reserves), it may potentially reduce harmful side effects to the athlete and to the relationship with the coach or scientist.

6.4.6 Future studies or question for exploration specific to this area of research

The current body of work has raised specific questions and inclinations towards future study designs. For example, despite emerging reviews (52, 120, 146, 147, 161) widely accepting decision-making skills as a key determining factor in pacing and performance, little empirical evidence exists. Such decisions of when and where athletes choose to change pace (i.e. changes in PO, cadence or gear) during self-paced exercise may provide further insight into how exercise is regulated in response to different internal and external stimuli.

In comparison, from a behavioural and thought process point of view, in a recent study by Stone et al. (171) the authors demonstrated video playback of each trial can provide additional evidence of when the participants choose to maintain or adjust the current work-rate. For example, when participants saw that the distance between the 'deception' avatar and their 'real-time' avatar began to widen, exercise intensity was increased as they attempted to match

the ‘deception avatar’ and mobilise or regain control of the TT. Expanding on Stone and colleagues work (171), involving participants in the video playback process immediately post-trial, in a qualitative interview based approach, may gain further insight into athletes thought processes at critical moments in the trial and whether they match up with performance data or are delayed as seen in the early stages of athletes blinded to the FiO_2 (78, 87).

Finally, research into complex psychological processes involved in pacing remains in its infancy. As a result, the pacing literature could further benefit from research exploring the role of pain modulation, placebo effects and mental skills training (i.e. arousal control, imagery or self-talk), to name but a few, to build on existing knowledge of the dynamic two-way conversation between incoming peripheral feedback and the brain to regulate the anticipated or current exercise bout (70, 129, 130, 173).

6.5 OPPORTUNITIES TO IMPROVE SELF-PACED EXERCISE PERFORMANCE: SUGGESTIONS FOR USE OF HYPEROXIA AND DECEPTION TO ACCESS AN EXERCISE RESERVE.

Based on findings from the current body of work, there is merit to suggest the provision of hyperoxia and deception, either separately or together, may be used as potential training modalities to improve subsequent normoxic performance. Logistically, coaches could allocate a two-week training period selecting one of the proposed training methods outlined on pages 196 to 197. Based on anecdotal feedback from participants who competed shortly after participating in Study Two or Three, the training block could be implemented three to four weeks out from competition, allowing two to one weeks to remove any residual fatigue from the hyperoxic sessions. However, these timeframes are to be are yet to be empirically confirmed and are speculative at present. A consideration before commencing hyperoxic, deception training sessions, or both, is the time between sessions due to the greater exercise capacity achieved in hyperoxia and deception trials which may induce greater levels of fatigue. Therefore, we suggest sessions should be scheduled at least 48 hours to allow adequate recovery to avoid residual fatigue between sessions. Additionally, preliminary testing in hyperoxia before commencing hyperoxic sessions is advised to ensure training intensities are matched or prescribed correctly to the artificially enhanced environment. If implemented appropriately, it is possible the provision of these two methods, separately or together may improve short-term normoxic trials or competitive events.

6.5.1 Suggested methods for use of hyperoxia and deception to access an exercise reserve:

i. Coaches exposing athletes to moderate levels of hyperoxia during TT's may alter task expectations, build belief and disrupt their athletes preconditioned pacing templates to improve subsequent normoxic performance. A visual example of how coaches and athletes might use self-paced (**Figure 6.4a**) or competitive (**Figure 6.4b**) hyperoxic TT's to improve performance is provided on page 196. It is worth mentioning athletes should be fully aware of the inspired oxygen to assist in building knowledge of the benefits of inspiring supplemented oxygen throughout these series of trials. A potential technique to achieve this is to provide instantaneous S_pO_2 data displayed on a computer monitor to accurately inform athletes they were not being deceived of the inspired oxygen content.

ii. In a different approach, a series of oxygen deception trials could be employed by coaches as an additional training method to educate their athletes on how an exercise reserve is often left in reserve during self-paced exercise, but if accessed can improve subsequent performances in normoxia. Furthermore, it appears crucial the order in which hyperoxic deception TT's are performed be considered to encourage athletes to deviate from their routine pacing template. Therefore, based on findings from Study Two, it appears a series of oxygen deception trials completed in order of: NORM (familiarisation trial), NORM-DEC, NORM, HYPER-DEC, HYPER and NORM-INFORM has the potential to build psycho-physiological belief in athletes such that performance may improve in subsequent performances. A visual example of the order described above is provided in **Figure 6.4c**.

iii. A final consideration for coaches to exploit athletes exercise reserve and improve short-term performance in normoxia is the provision of negative performance

deception. This type of deceptive feedback used in normoxia or hyperoxia may be a beneficial training tool for athletes to access their energy reserves by eliciting a subtle upregulation in exercise intensity but not too great to be intolerable. An example of how negative deceptive feedback can be used in normoxia (**Figure 6.5**) or hyperoxia (**Figure 6.6**) is suggested on page 197. Like hyperoxic self-paced and competitive TT's, athletes in hyperoxic competitive-deception trials should be fully aware of the inspired oxygen to assist in building knowledge of the benefits of inspiring supplemented oxygen throughout the series of trials.

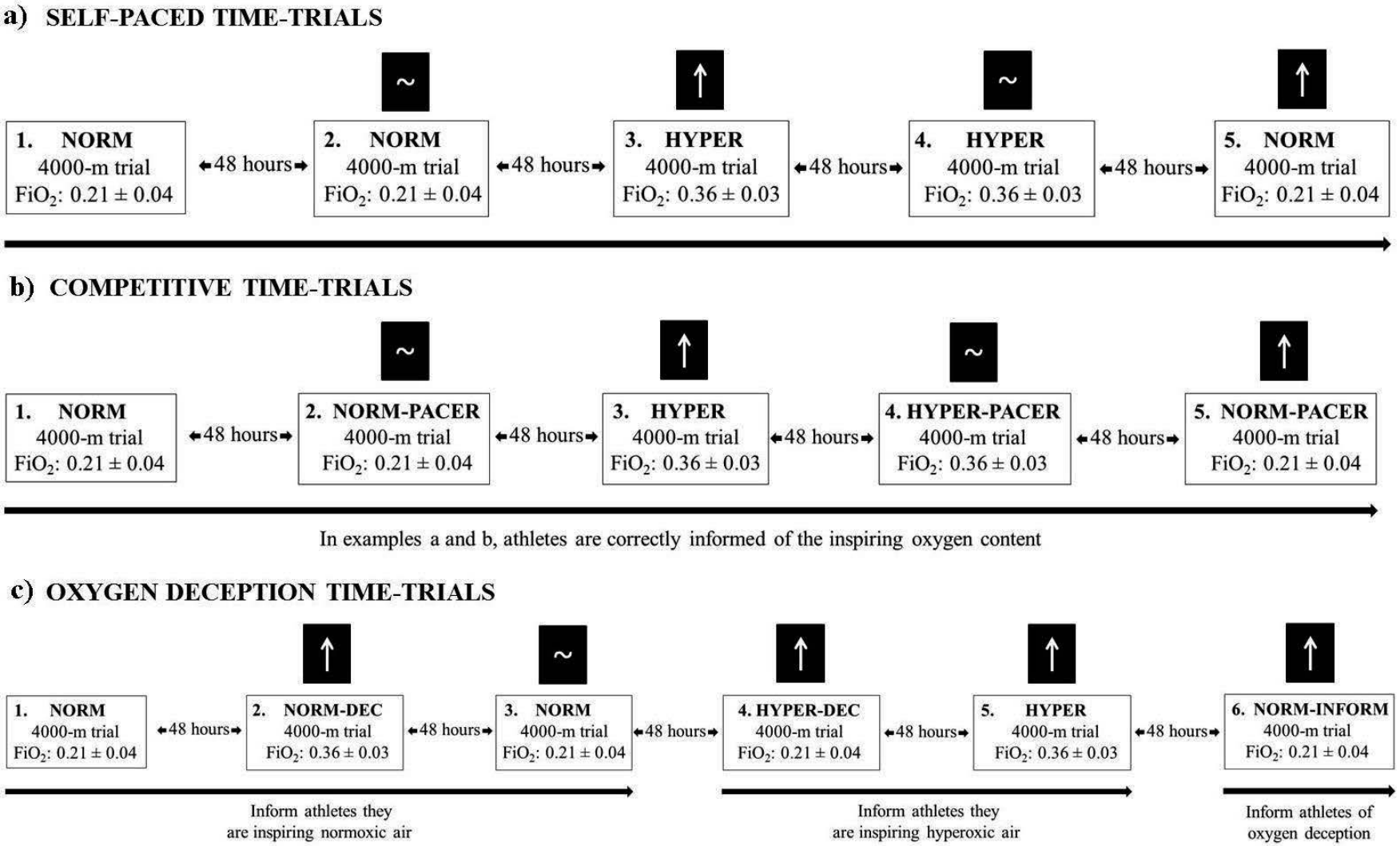


Figure 6.4 Suggestions for use of hyperoxia in self-paced (**a**), competitive (**b**) and oxygen deception (c) time-trials; ↑ indicates an expected MPO increase while ~ indicates no change compared to NORM.

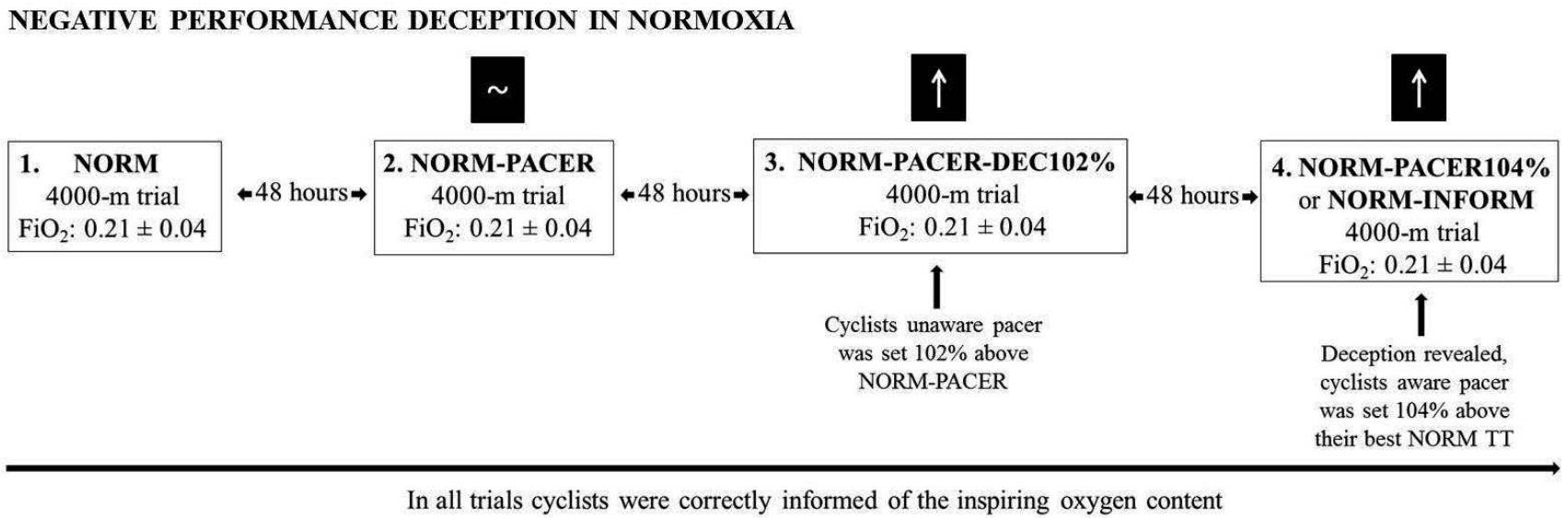


Figure 6.5 Suggestions for use of negative performance deception in normoxia; ↑ indicates an expected MPO increase while ~ indicates no change compared to NORM.

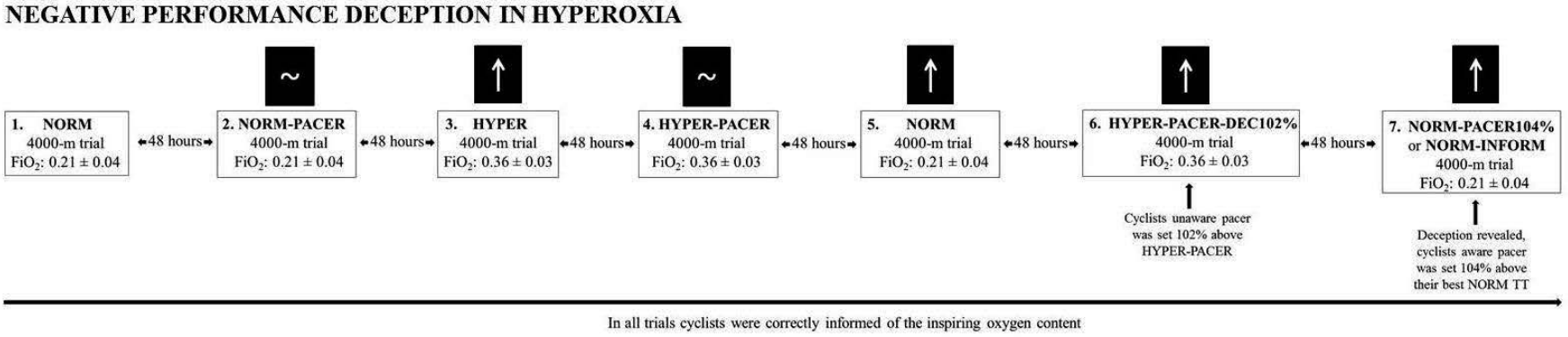


Figure 6.6 Suggestions for use of negative performance deception in hyperoxia; ↑ indicates an expected MPO increase while ~ indicates no change compared to NORM.

6.6 CONCLUSION

The aim of this thesis was to examine how environmental and performance feedback manipulations could affect exercise regulation and performance in trained cyclists. The achievement of the objectives in this thesis has provided an examination of the body's response to exercise from different environmental and feedback manipulations, and specifically, how exposure to hyperoxic air and performance deception may influence exercise regulation in trained cyclists. The novel findings of this research were: i) self-paced TT's undertaken in hypoxia or heat-stress had no effect on cycling compared with normoxic or cool conditions until the middle section of trials where exercise intensity was downregulated (Study One); ii) provision of negative deceptive feedback may enable upregulation of MPO during the middle stages of a trial improving overall trial performance (Study One); iii) performance improvements in a previous negative deceptive feedback trial may be retained in a subsequent TT after deception is revealed (Study One); iv) cycling performance improves in hyperoxia, and mechanisms are likely physiological, however, improvement in a deception trial suggests a placebo effect might be present. (Study Two); v) a particular sequence of oxygen deception trials might build psycho-physiological belief to improve performance in a subsequent normoxic trial (Study Two); vi) further performance improvement can occur when receiving negative deceptive feedback in hyperoxia which indicates an exercise reserve still exists in hyperoxia (Study Three); vii) performance improvements after negative performance deception in hyperoxia can be retained in a subsequent normoxic TT after deception is revealed (Study Three); and viii) no harm or decrement to performance was observed by exposing cyclists to hyperoxic air or performance deception (Study Two and Three). These findings contribute to our knowledge of how exercise is modulated and offer mechanistic understanding of how trained cyclists can change

their routine pacing strategy to enhance performance from exposure to increased FiO_2, oxygen deception trials and negative performance deception.

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APPENDIX 1: PUBLICATION 1 – STUDY ONE

Author's Note: Full Text of Study One is provided from pages 214 to 237.

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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

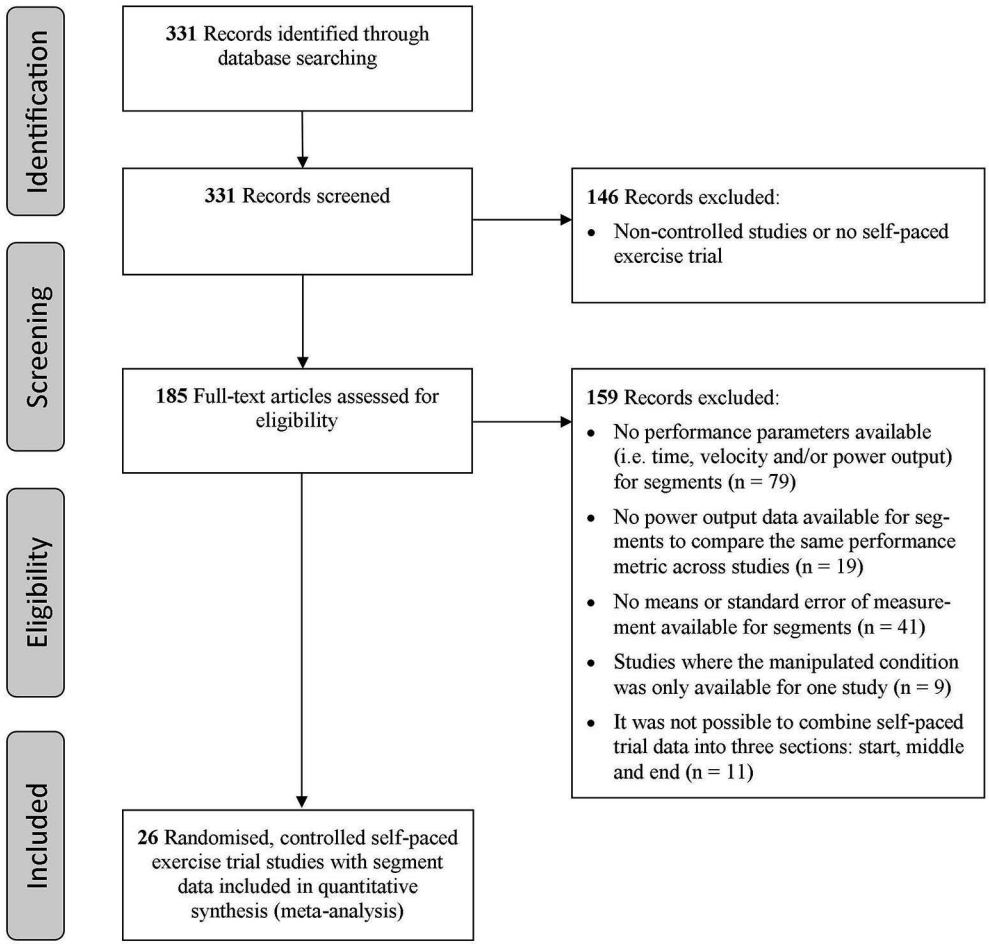

FIGURE 1 | Flow chart summary of the study selection process.

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

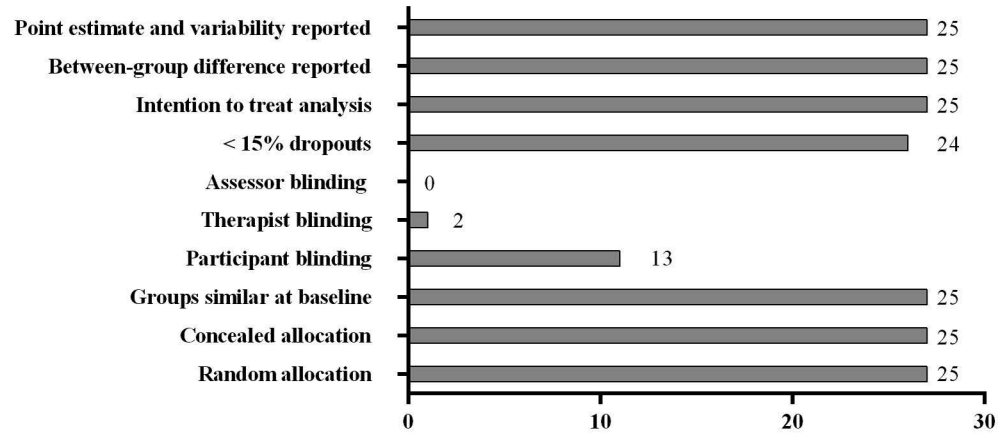

FIGURE 2 | Number of studies meeting individual PEDro [Physiotherapy Evidence Database] criteria.

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

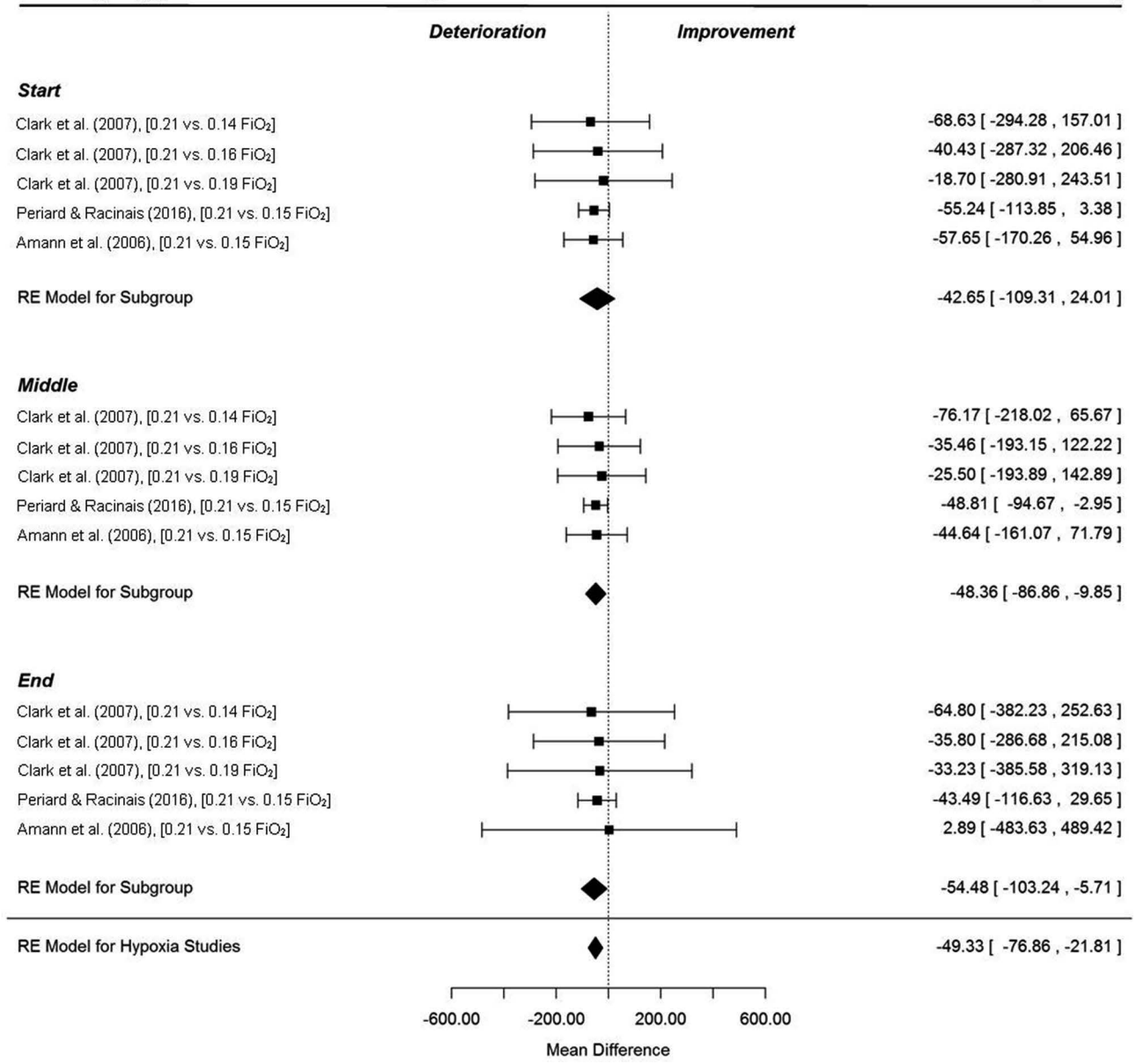

FIGURE 3 | Forest plot for hypoxia meta-analysis illustrating power output during start, middle and end sections compared to normoxia trials. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the 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, $p = 0.414$)

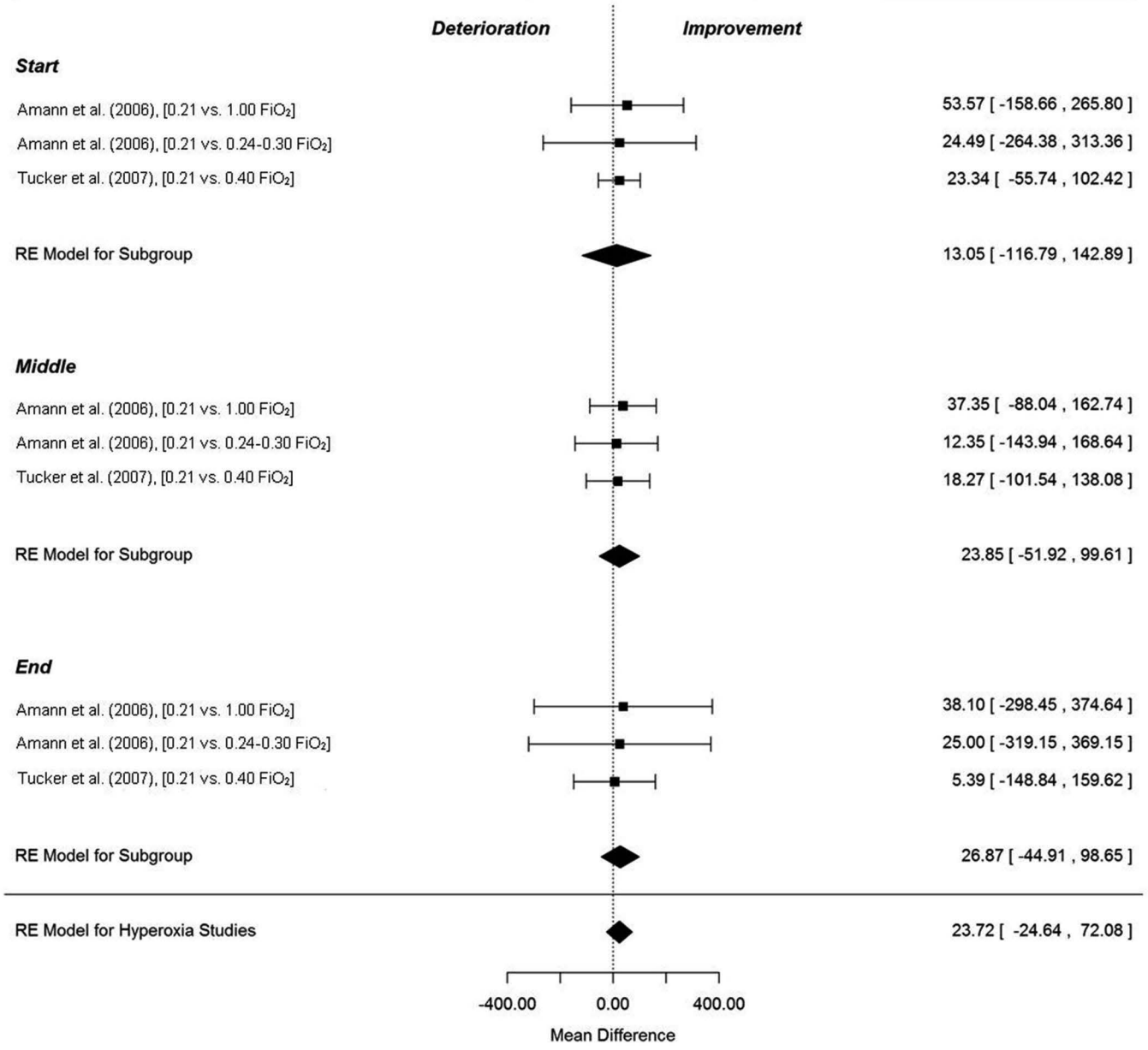

FIGURE 4 | Forest plot for hyperoxia meta-analysis illustrating power output during start, middle and end sections compared to normoxia trials. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within 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 =

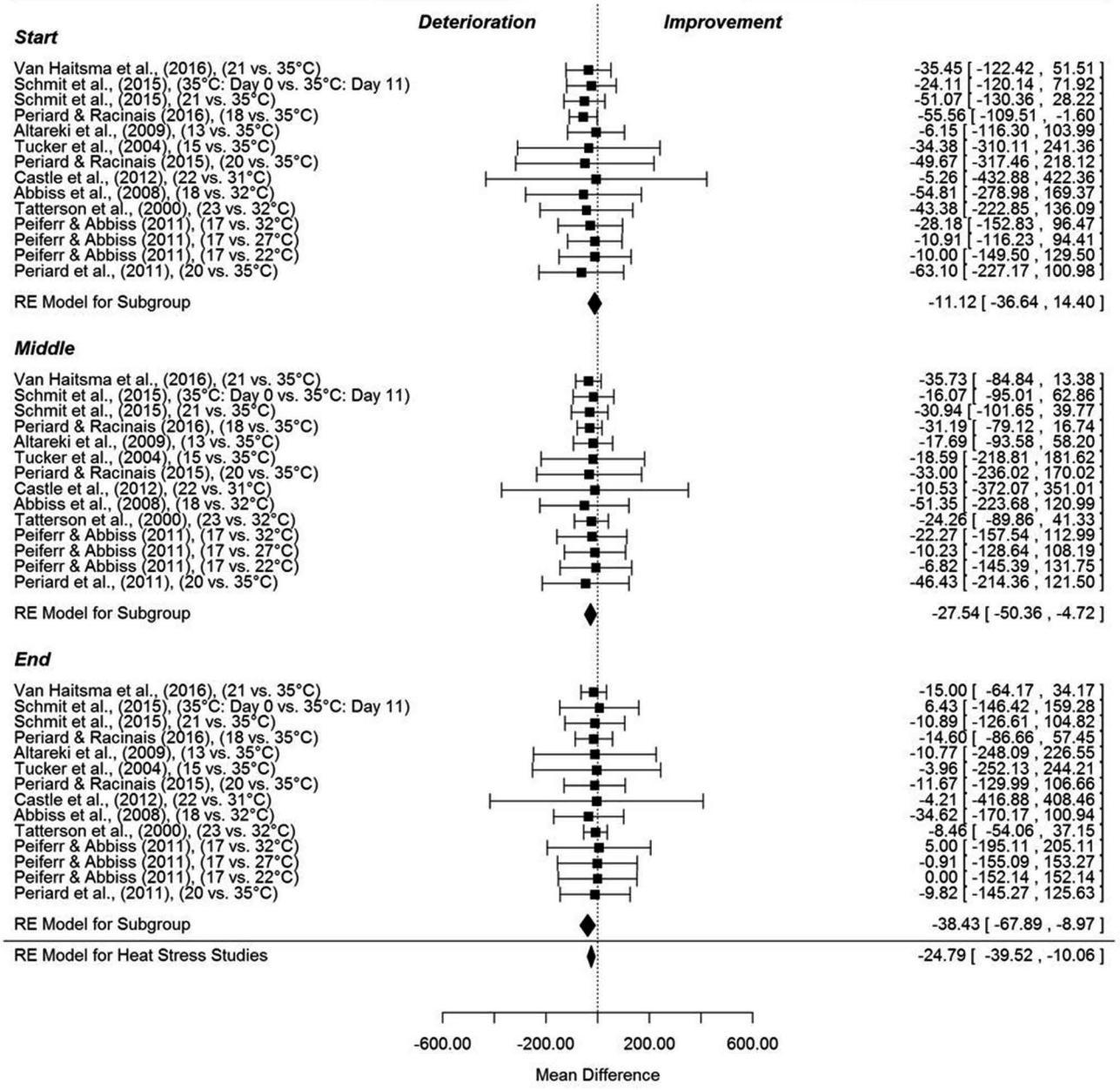

FIGURE 5 | Forest plot for heat-stress meta-analysis illustrating power output during start, middle and end sections compared to temperate trials. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within 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

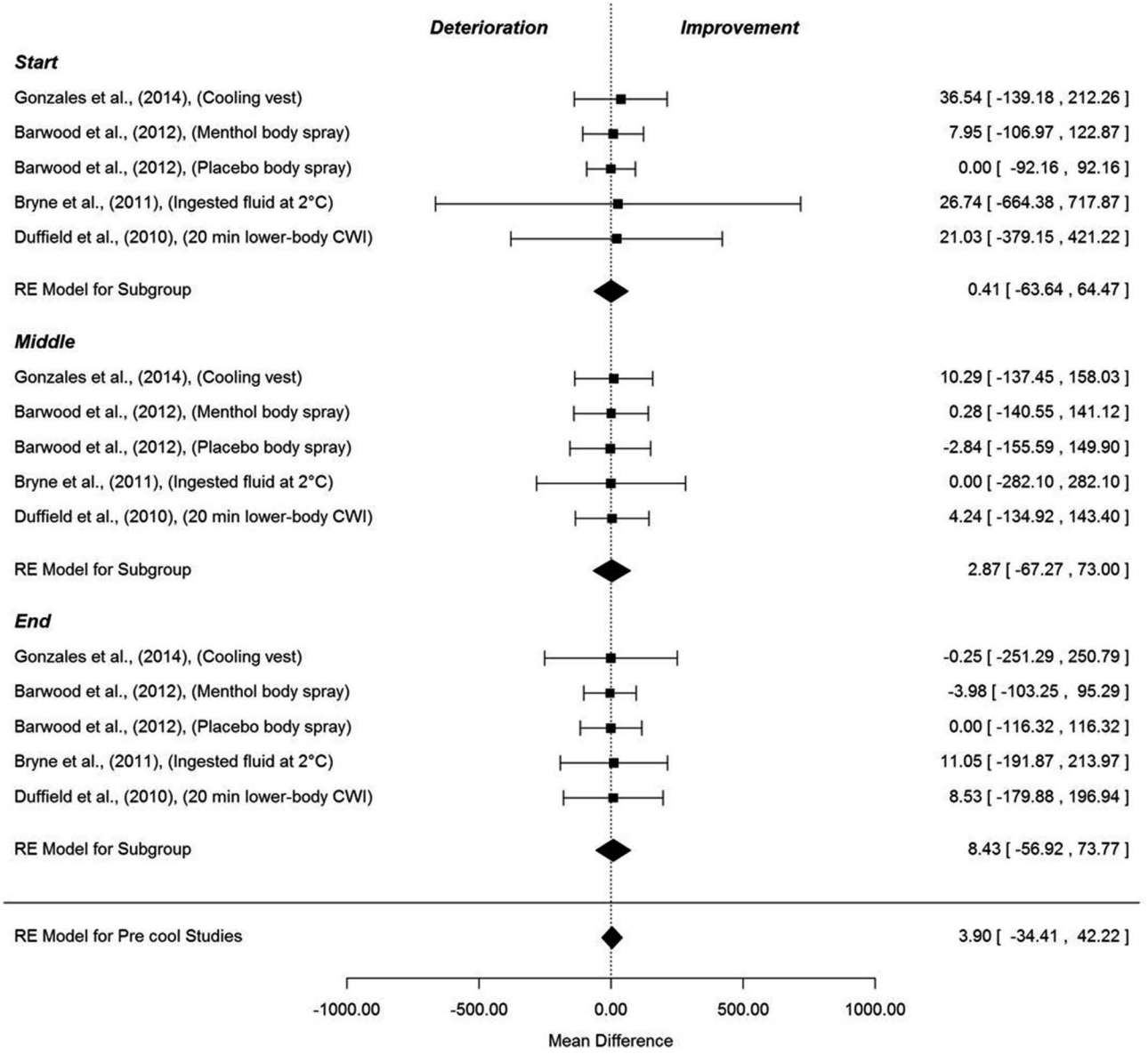

FIGURE 6 | Forest plot for pre-cooling intervention meta-analysis illustrating power output during start, middle and end sections power output for start, middle and end sections compared to no intervention. Squares represent individual study mean difference and the lines represent 95% CIs. The size of 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

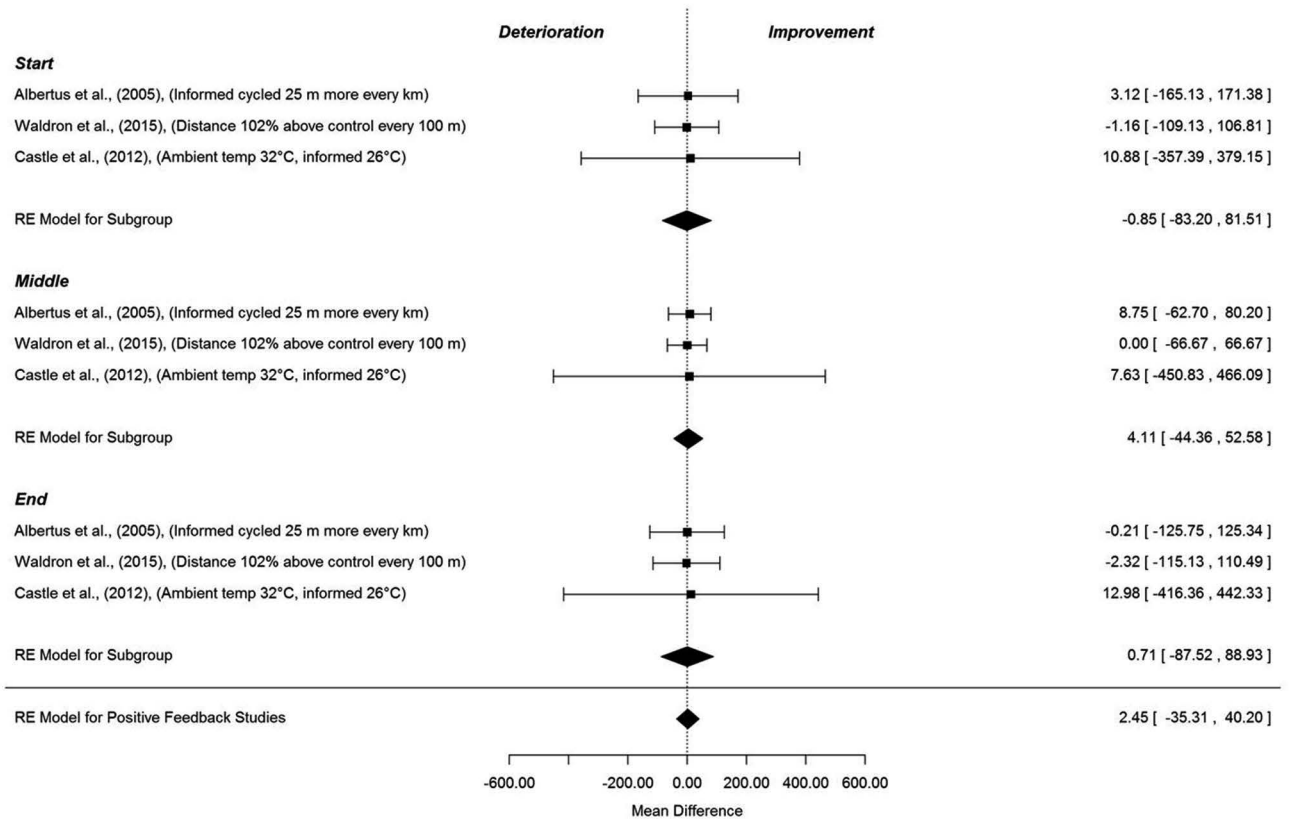

FIGURE 7 | Forest plot for positive deceptive feedback meta-analysis illustrating power output during start, middle and end sections power output for start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size 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

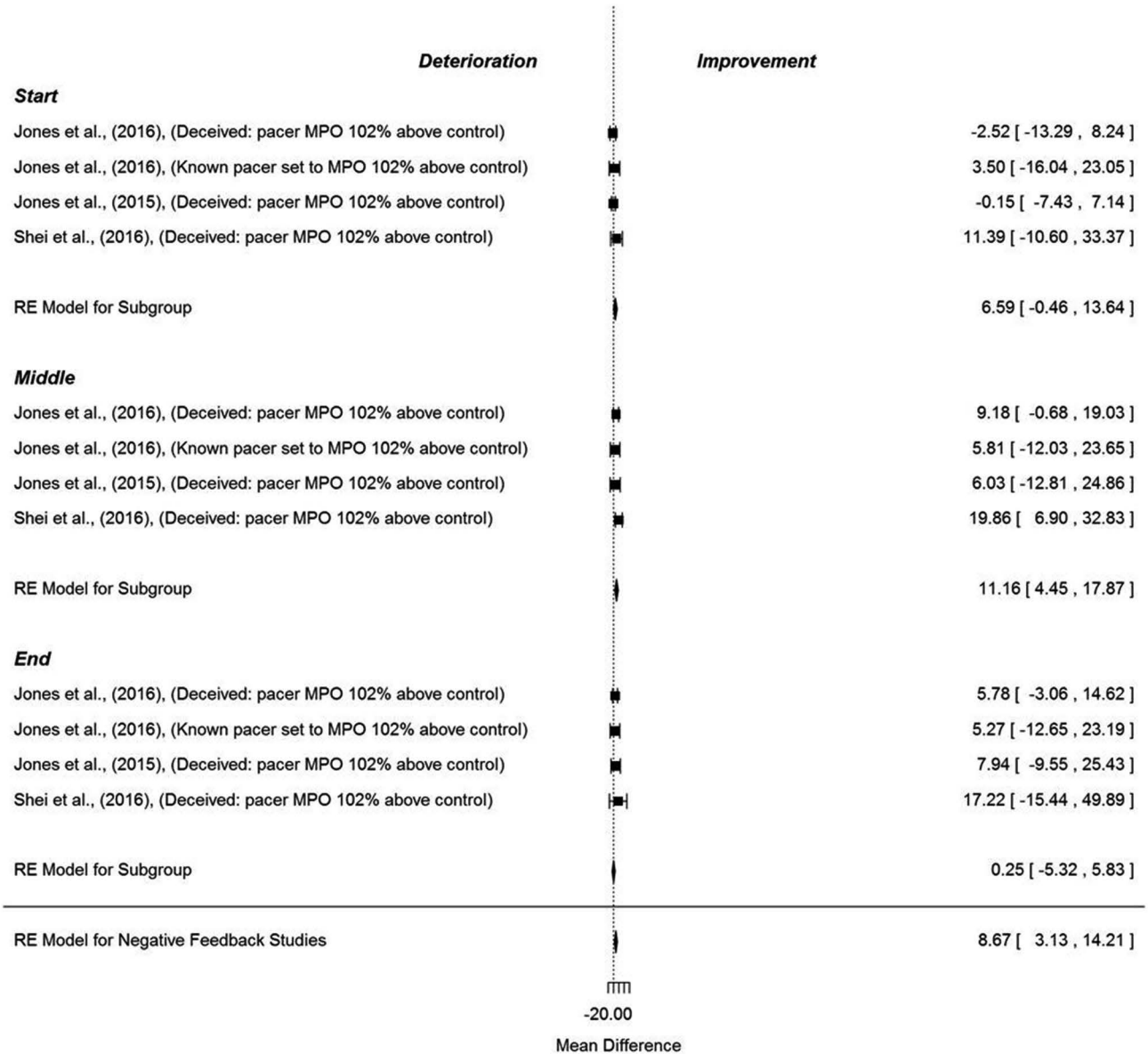

FIGURE 8 | Forest plot for negative deceptive feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the 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

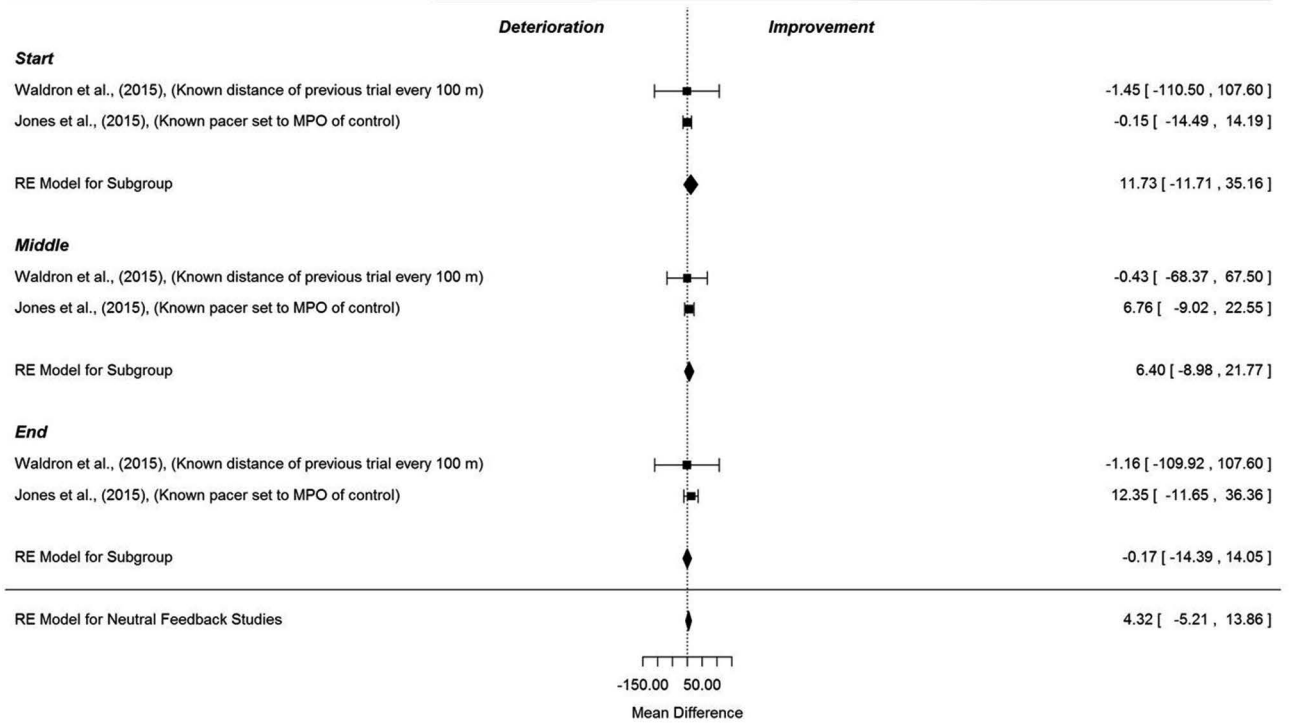

FIGURE 9 | Forest plot for neutral feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within 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

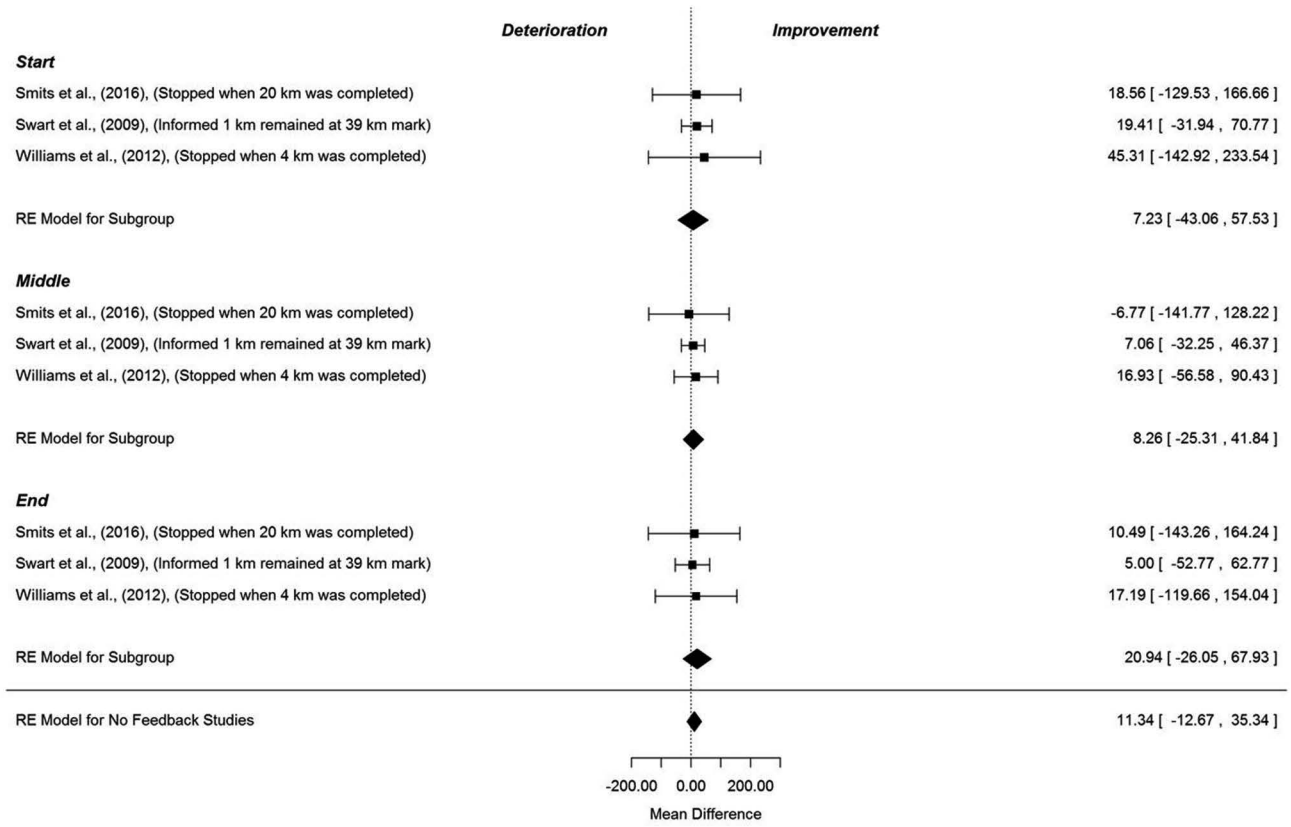

FIGURE 10 | Forest plot for no feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within 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

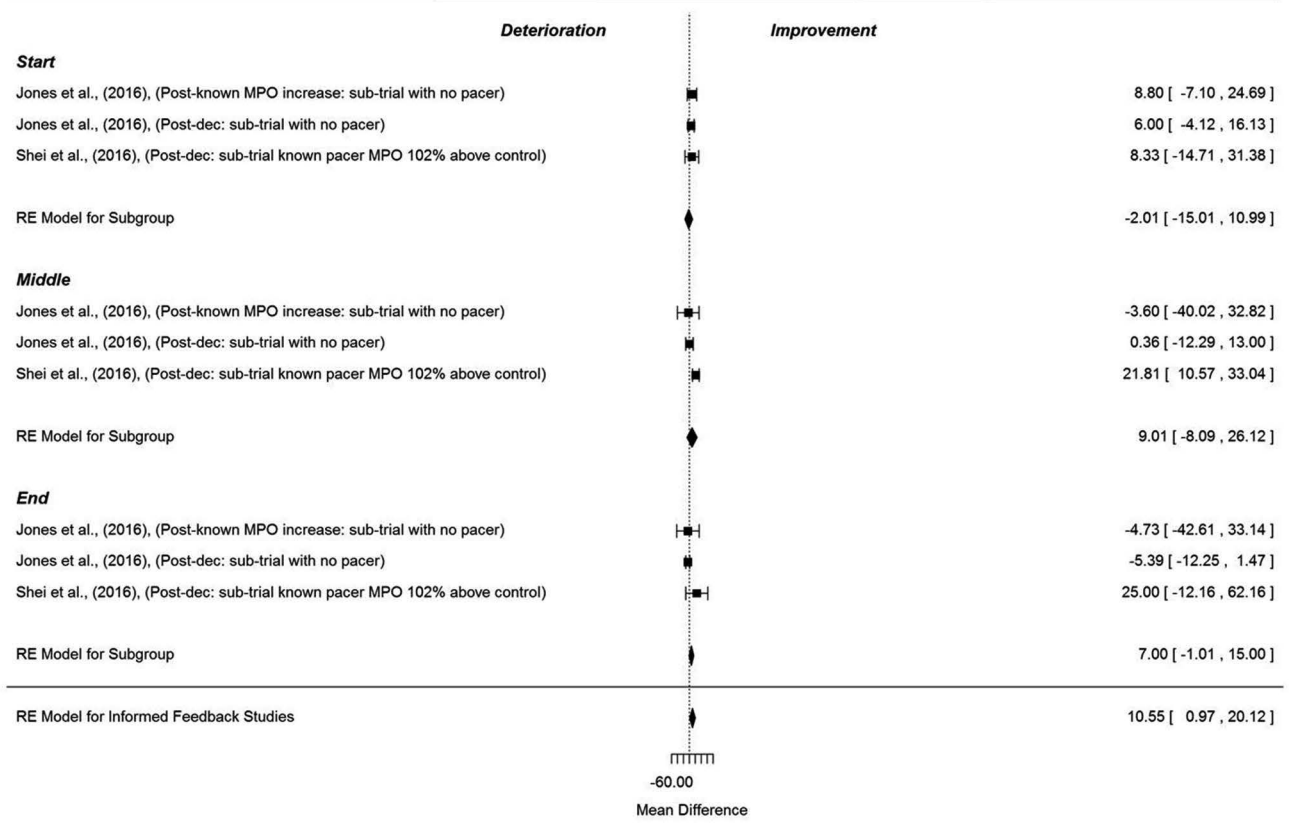

FIGURE 11 | Forest plot for informed feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study 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 ≤ 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

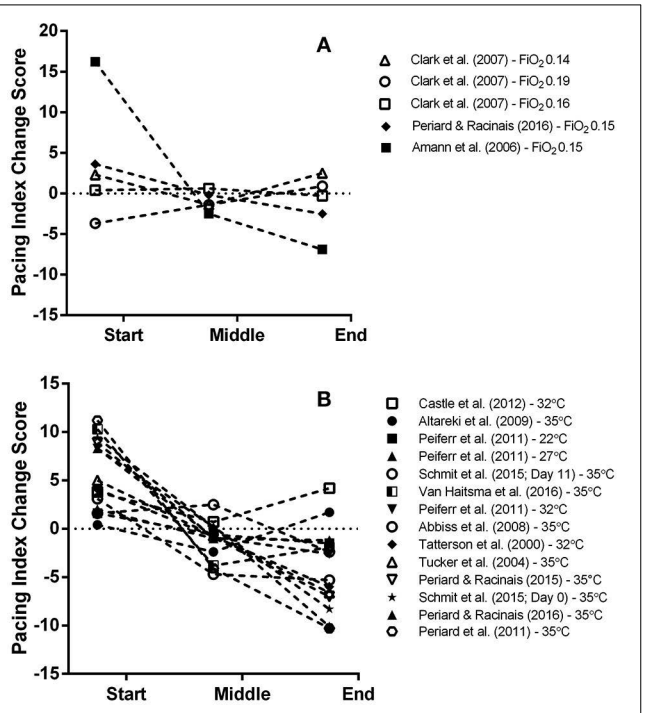

FIGURE 12 | Exploratory graphical analysis of the pacing index change score difference between control and hypoxic condition (A) and control and heat-stress condition **(B)** for each individual segment. For an explanation of how the pacing index change score was calculated please refer to section Data 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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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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APPENDIX 2: INSPIRING HYPEROXIA DURING EXERCISE FACTSHEET – STUDY TWO & THREE

Author's Note: Participant factsheet is provided from pages 240.

Participant Information

UCRISE & AIS – Hyperoxia Study

Overview

- The aim of the study is to investigate the effect breathing oxygen enriched air, known as hyperoxia, has on pacing and performance during 4,000m time-trials for trained cyclists.
- During the study you will complete six 4-km time-trials.
    - 3 x normoxic trials
    - 3 x hyperoxic trials
- In each trial you will be blind to all performance feedback except for elapsed distance.
- This will ensure you give your best effort regardless of how you feel or pace yourself on the day.

Normoxic trials

Purpose of completing three normoxic trials is to:

1. Familiarise you with the testing protocol.
2. Understand how you perform under normal environmental conditions.

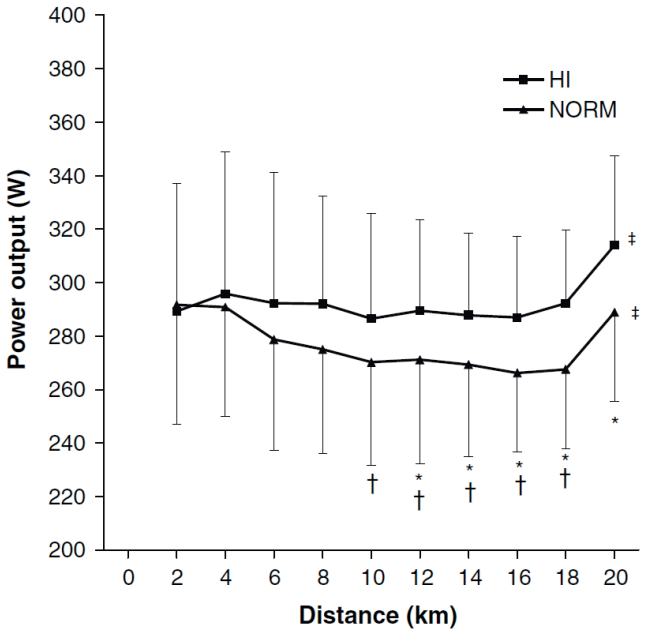
Hyperoxic trials

Purpose of completing three normoxic trials is to:

1. Collect data on how you perform in hyperoxia.
2. Determine the effects of hyperoxia on the contribution of aerobic and anaerobic energy systems, energy expenditure and pacing during 4-km time-trials.

What you can expect?

- Research has shown that hyperoxia can significantly improve cycling performance. See results from Tucker et al. (2007) study:



Average power output for 2 km intervals during time-trials in HI and NORM. Values are means ± SD for 11 subjects.

- From our pilot testing at the AIS with world class team pursuit cyclists we have found similar results and expect a comparable trend of increased power output during your hyperoxic trials.

Research Outcome

- It is expected the results from this project will provide direction and insight for coaches and sport scientists to use hyperoxia as a training modality in the lead up to Rio 2016.

APPENDIX 3: SRM POWER METER CALIBRATION – STUDY THREE

Author's Note: The following information outlines how the SRM power meter was calibrated in Study Three:

- The dynamic calibration rig used in this study was calibrated using four accredited masses up to 11.39 kg using the most popular professional version of the SRM power meters (i.e., four strain gauges), mounted to a VelotronDynafit Pro cycle ergometer (RacerMateInc, CITY, WA, USA).
- As described previously by Abbiss et al. [ref], the 'calibration rig' is a torque reaction measuring device. A rotational mechanical drive is delivered to the ergometer via a double universal joint to the axis of rotation of the pedal shaft.
- The calibration rig quantifies the reaction torque (N·m) using a calibrated load cell positioned 1 m from the fulcrum point, and the angular velocity (Rad·s-1) using a precision period measurement with crystal-based timing (10 MHz oscillator).
- Power is then calculated as the product of torque and angular velocity.
- Before and after each testing protocol, the load cell was calibrated using a National Association of Testing Authorities certified mass (11.39 kg).
- The SRM power meter (Ingenieurbüro Schoberer, Jülich, Germany) is a power monitoring system that calculates the power delivered to the bottom bracket by measuring the torque applied to the cycle crank along with the crank cadence. The scientific SRM used in Study Three measured torque via the deformation of eight in-parallel strain gauges that are located within the crank, while cadence is detected using a 'reed contact' switch.

- Prior to each test and after a 11-minute warm-up (describe in section 5.3.3 under heading *4000-m time-trial protocol* for *Experimental Procedures*) the zero off set of the SRM power meter was checked and set in accordance with the manufacturer's instructions.