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Article Title: The Potential to Change Pacing and Performance During 4000-m Cycling Time Trials Using Hyperoxia and Inspired Gas-Content Deception

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The potential to change pacing and performance during 4000-m cycling time trials using hyperoxia and inspired gas-content deception.

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Abstract:

Purpose: Determine if a series of trials with fraction of inspired oxygen (FiO_2) content deception could improve 4000-m cycling time-trial (TT) performance. **Methods:** Fifteen trained male cyclists (mean \pm SD: body mass 74.2 ± 8.0 kg; peak oxygen uptake $62 \pm 6 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) completed six, 4000-m cycling TTs in a semi-randomised order. After a familiarisation TT, cyclists were informed in two initial trials they were inspiring normoxic air (NORM, FiO_2 : 0.21), however in one trial (deception condition) they inspired hyperoxic air (NORM-DEC, FiO_2 : 0.36). During two subsequent TTs, cyclists were informed they were inspiring hyperoxic air (HYPER, FiO_2 : 0.36), but in one trial normoxic air was inspired (HYPER-DEC). In the final TT (NORM-INFORM) the deception was revealed, and cyclists were asked to reproduce their best TT performance while inspiring normoxic air. **Results:** Greater power output and faster performances occurred when cyclists inspired hyperoxic air in both truthful (HYPER) and deceptive (NORM-DEC) trials compared to NORM ($P < 0.001$). However, performance only improved in 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:** 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.

Introduction:

Pacing strategies are often defined as pre-determined 'templates' which are built through prior experience and recalled for future performances.¹ 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.² Recently, researchers have manipulated athletes expectations and beliefs regarding their abilities to examine how performance can be enhanced through access of reserve capacities.^{3,4} To achieve this, deceptive information is provided, therein manipulating perceptual, environmental or performance feedback via non-invasive and practical methods.⁴ The purpose of such deception is to create uncertainty within the pacing template, causing athletes to deviate from their routine strategy.³ However, results from a recent meta-analysis suggest changes in pacing and performance is largely dependent on the type of deception received.⁵

Studies have delivered deceptive information by informing athletes they are performing better (positive) or worse (negative) via inaccurate performance information.⁵ Negative deceptive feedback, specifically performance deception, appears the most effective form of deception to elicit performance improvements.⁵ 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.⁵⁻⁸ Interestingly, performance improvements in a negative performance deception trial, may also be retained in a subsequent TT once deception is revealed.^{6,8} 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.⁸ However, whether deception regarding environmental conditions can elicit similar responses is largely unknown.

Environmental deception is designed to change athletes task expectations in an attempt to 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.⁹ 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.⁵ Previous studies blinding athletes to the FiO_2 indicate even slight manipulations are sufficient to influence cycling performance.^{10,11} Specifically, self-paced cycling completed in hyperoxia (FiO_2 : > 0.21) has demonstrated performance improvements by 3–14% compared with normoxic trials.^{12–15} However, to our knowledge, no current research describes the effects of FiO_2 deception on athletic performance. Therefore, whether knowledge of inspiring hyperoxic air encourages an upregulation of exercise intensity compared to normoxia requires further exploration. Moreover, whether a series of FiO_2 deception trials can build psycho-physiological belief in cyclists to improve performance in a subsequent normoxic trial 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 content and belief (though deception) might change pacing and performance during 4000-m cycling TTs. Collectively this information will further our understanding of how hyperoxia and deceptive information alters trained athletes pacing and further, determine if these techniques could be used in training to enhance performance.

Methods:

Participants:

Fifteen trained male cyclists (mean \pm SD: stature 181 ± 6.7 cm; body mass 74.2 ± 8.0 kg; peak oxygen uptake ($\dot{V}O_{2\text{peak}}$) 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 24 hours preceding each test. The study was approved by the University of Canberra Research Ethics Committee.

Experimental design:

Cyclists completed six 4000-m cycling TTs, 48 hours apart over two weeks (Figure 1). In trials one to three, cyclists were informed they were in normoxia (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 hyperoxic deception (HYPER-DEC). However, in HYPER-DEC cyclists were inspiring normoxic air. ‘NORM’ and ‘HYPER’ trials were completed in a double-blinded and randomised order, meaning cyclists completed trials in one of four possible orders: HNNH (i.e. NORM-DEC, NORM, HYPER, HYPER-DEC), HNNH, NHHN and NHNH. A sixth and final trial was then completed, where all cyclists were informed they had been deceived, and were asked to reproduce their best performance whilst in normoxia (NORM-INFORM, FiO_2 : 0.21 ± 0.04). After the final trial participants were asked whether they suspected they had been deceived of the FiO_2 across trials. Participants reported they were unaware of the FiO_2 deception and could not correctly identify inspired gas contents for specific trials.

Procedures

Preliminary Testing:

All TTs were completed on a VelotronDynaFit Pro cycle ergometer (RacerMateInc, CITY, WA, USA) with power output (PO) sampled at 1 Hz.¹⁶ 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.¹⁷ The highest $\dot{V}O_2$ averaged over a 30 second period was defined as $\dot{V}O_{2peak}$. Maximal aerobic power was determined as the final completed stage plus the proportion of any uncompleted stage attempted during the GXT.¹⁸ 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 gas mixture of the specific trial was inspired. The purpose of the wash in phase was to acclimate cyclists to the administered 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 and passive recovery, respectively, before commencing the TT.¹⁹ 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 derived using the following equation.²⁰

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

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 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 ($\sim 20\text{-km}\cdot\text{h}^{-1}$). 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.²¹

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, produced by mixing ambient air with medical grade oxygen (O_2 concentration 100%, BOC Gases and Gear, Canberra, Australia) for hyperoxic trials, 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: $\text{FiO}_2 0.21 \pm 0.04$; 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).

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.²² Briefly, 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 to elicit greater total force production as previously suggested.²¹ 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, abraded and cleaned, before positioning two electrodes (inter-electrode distance of 30-mm) on the midline belly of the muscle.²³ An electronic goniometer (ADInstruments, NSW, Australia) was positioned and secured on the lateral side of the right knee to set 100° knee flexion in the pre- and post- MVC, and secondly to trigger the corresponding sEMG at 100° knee flexion ($99.7 \pm 0.1^\circ$) during each downward pedal stroke during TTs. 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.²³ 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.²⁴ 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.²⁵

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.^{26,27} 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 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.²⁸ Statistical significance was accepted at $P < 0.05$. Data are reported as mean estimates and 95% confidence intervals (CI).

Results:

Time-trial performance and pacing:

No Trial Order by Condition by Segment interaction was found for MPO ($F_{135,649} = 0.806$, $P = 0.939$). However, Trial Order by Condition ($F_{15,649} = 3.216$, $P < 0.001$, Figure 2) and Trial Order by Segment ($F_{27,649} = 4.680$, $P < 0.001$, Figure 3a) interactions for MPO were significant. Similarly, a Trial Order by Condition interaction ($F_{15,649} = 2.616$, $P < 0.001$) was found for performance time, however, Trial Order by Segment was not significant ($F_{27,649} = 0.637$, $P = 0.924$). 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 2d). Additionally, across all groups, significantly greater MPOs and faster performances were recorded in hyperoxia compared to NORM, in both truthful (HYPER: 31 W, 9%) and deceptive trials (NORM-DEC: 25 W, 8%,

$P < 0.001$, Figure 2). Finally, a significant Trial Order by Condition ($F_{15,649} = 6.276$, $P < 0.001$, Figure 4) interaction was found for cadence, although Trial Order by Segment was not significant ($F_{27,649} = 0.104$, $P = 0.104$).

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 2). However, a significant Trial Order by Segment interaction was found ($F_{27,649} = 5.461$, $P < 0.001$), where a faster starting strategy for all TTs in order HNNH and an end spurt initiated earlier in order HNNH were found compared to other groups (Figure 3b).

Heart rate:

Trial Order by Condition ($F_{15,609} = 9.241$, $P < 0.001$, Figure 4) and Segment ($F_{27,609} = 3.658$, $P < 0.001$) 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 4c). In contrast, the HR response was similar across all other conditions and orders.

Muscle activation during time-trials:

Trial Order by Condition interactions for iEMG ($F_{15,566} = 17.168$, $P < 0.001$) and ratio of PO to iEMG were significant ($F_{15,566} = 20.105$, $P < 0.001$). 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 5).

Voluntary force production and muscle activation:

No significant Time by Trial Order by Condition interaction was found for peak force during the MVC ($F_{12,96} = 0.802$, $P = 0.647$). Similarly, the interaction between ratio of muscle

activation for peak force was not significant for Time by Trial Order by Condition ($F_{12,95} = 0.651$, $P = 0.793$). However, a significant time effect was found, where peak force declined significantly one minute post-TTs ($P < 0.001$), associated with a significant reduction in post-trial peak force to sEMG ratio ($P < 0.001$, Figure 6).

Motivation and emotion responses:

There was no significant Trial Order by Condition for success ($F_{15,55} = 0.552$, $P = 0.898$) or intrinsic motivation ($F_{15,55} = 0.879$, $P = 0.510$). No significant Trial Order by Condition was found in the Sports Emotion Questionnaire post TTs for: anxiety ($P = 0.655$), dejection ($P = 0.310$), excitement ($P = 0.390$), anger ($P = 0.514$) and happiness ($P = 0.210$).

Discussion:

The present study demonstrated FiO₂ content deception trials were not sufficient to improve performance in a subsequent normoxic cycling TT. However, greater PO and faster performances occurred when cyclists inspired hyperoxic air in both truthful (HYPER) and deceptive (NORM-DEC) trials compared to NORM ($P < 0.001$). Furthermore, cyclists who completed trials in HNNH improved 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 TTs completed on a cycle ergometer (< 6 W or 0.7 – 1.4 seconds),²⁹ therefore, there was no decrement in performance irrespective of trial order.

Previous studies have suggested if cyclists can improve performance in a normoxic informed trial, it might indicate they had accessed an exercise reserve above their perceived maximal capacity.⁶⁻⁸ 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 2c), which was associated with a significantly greater HR response (Figure 4c) similar to earlier reports after deception was revealed.⁷ By comparison, previous reports have suggested athletes can access reserve capacities up to 2% for MPO before the exercise intensity becomes intolerable.⁸ One possible explanation is knowledge of previous trial performance, compared to perception of effort during NORM-INFORM, may have influenced the cyclists belief to upregulate exercise intensity.⁴ However, this is somewhat speculative since we found no significant change in pre-motivation or post-trial emotional responses across all trial orders and measures of cyclist's belief, competency or expectation were not quantified before the final trial.³⁰ 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 developed over this order leading up to NORM-INFORM. A further explanation for the observed improvement in NORM-INFORM might be that cyclists were in hyperoxia in their preceding TT. Although this is unlikely as no improvement was observed in NORM-INFORM when cyclists completed trials in order NHHH. 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 TTs in order HNNH compared to NHHH, suggesting the FiO_2 in preceding TTs may have altered pacing in subsequent trials as previously reported.³¹ It is possible improved performance in NORM-INFORM by those cyclists completing trial order HNNH was the result of a training effect.²⁹ However, given performance improvements in NORM-INFORM were evident only for these participants this explanation seems unlikely.

An additional aim was to examine whether environmental deception changed pacing during the deception trials themselves. Cyclists completing 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 normoxic air when believing they were in

hyperoxia, suggesting this belief influenced their performance. Therefore, it is possible improved TT performance in hyperoxia is due to physiological and potentially a psychological enhancement (placebo effect). Conversely, a negative experience in HYPER-DEC in other trial orders may have induced a nocebo effect that manifested in subsequent performances.³⁰ However, this is unlikely given no participant indicated awareness of deception nor could they correctly identify the administered gas content. Consequently, order HNNH may have been sufficient to disturb cyclists routine pacing template, causing the current work-rate to be upregulated despite being in normoxia. In comparison, cyclists maintained greater MPO in NORM-DEC compared to NORM across all orders. Therefore, believing they were in normoxia did not appear to inhibit the benefits of inspiring hyperoxic air, contrary to many models and theories on pacing which emphasise the importance of prior experience overcoming peripheral feedback.¹ This finding is in agreement with previous reports, where cyclists will adjust exercise intensity despite being unaware of inspiring hyperoxic air, likely a result of associated mechanisms responding to sensory feedback from the periphery.¹⁰⁻¹⁵ Therefore in the present study, it is possible feedback in the periphery provided a sufficient stimulus to overcome any pre-existing pacing template;¹⁵ for instance, blood chemoreceptors,¹⁴ group III-IV muscle afferents¹⁵ or metabolic milieu.¹³ 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 .

The current investigation supports previous studies which have found increased FiO_2 improves self-paced exercise.¹²⁻¹⁵ However, irrespective of trial order, a novel finding is the benefit of 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 $\dot{V}\text{O}_2$ and faster $\dot{V}\text{O}_2$ kinetics,¹⁴ oxygen availability at skeletal muscles and reduced biochemical

and physiological disturbances to homeostasis.¹² For instance, in hyperoxia cerebral oxygenation is increased reducing levels of fatiguing metabolites at working musculature,¹³ often limiting factors for exercise in normoxia.¹² 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.¹²⁻¹⁵

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.² 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, suggesting hyperoxia did not decrease the development of peripheral muscle fatigue.¹² However, visual 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.²⁹ Nevertheless, changes in iEMG amplitude can still provide a practical reflection of changes in muscle activation during self-paced exercise.¹⁶

Practical Applications:

- Hyperoxic and oxygen deception TTs have the potential to build belief and improve performance in subsequent normoxic trials or competitive events.
- The order of hyperoxic and deception TTs may encourage athletes to deviate from their routine pacing template.

- An appropriate pacing strategy should be considered when altering the FiO_2 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.

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.

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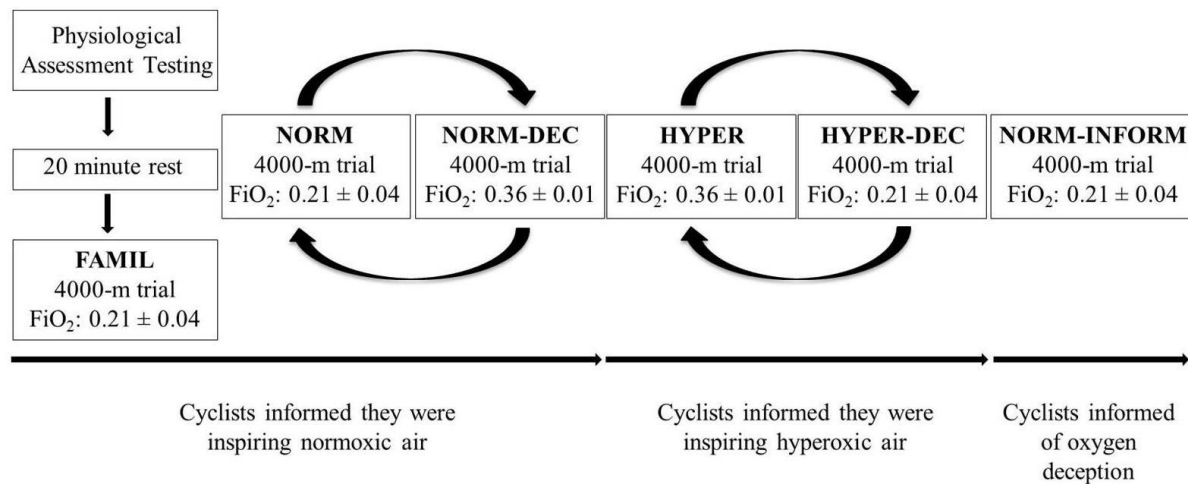


Figure 1. Study design, including actual and informed FiO_2 for each trial. Randomised trials are indicated by black arrows. Both the cyclists and researcher (who conducted the experimental trials) were blinded to the actual FiO_2 .

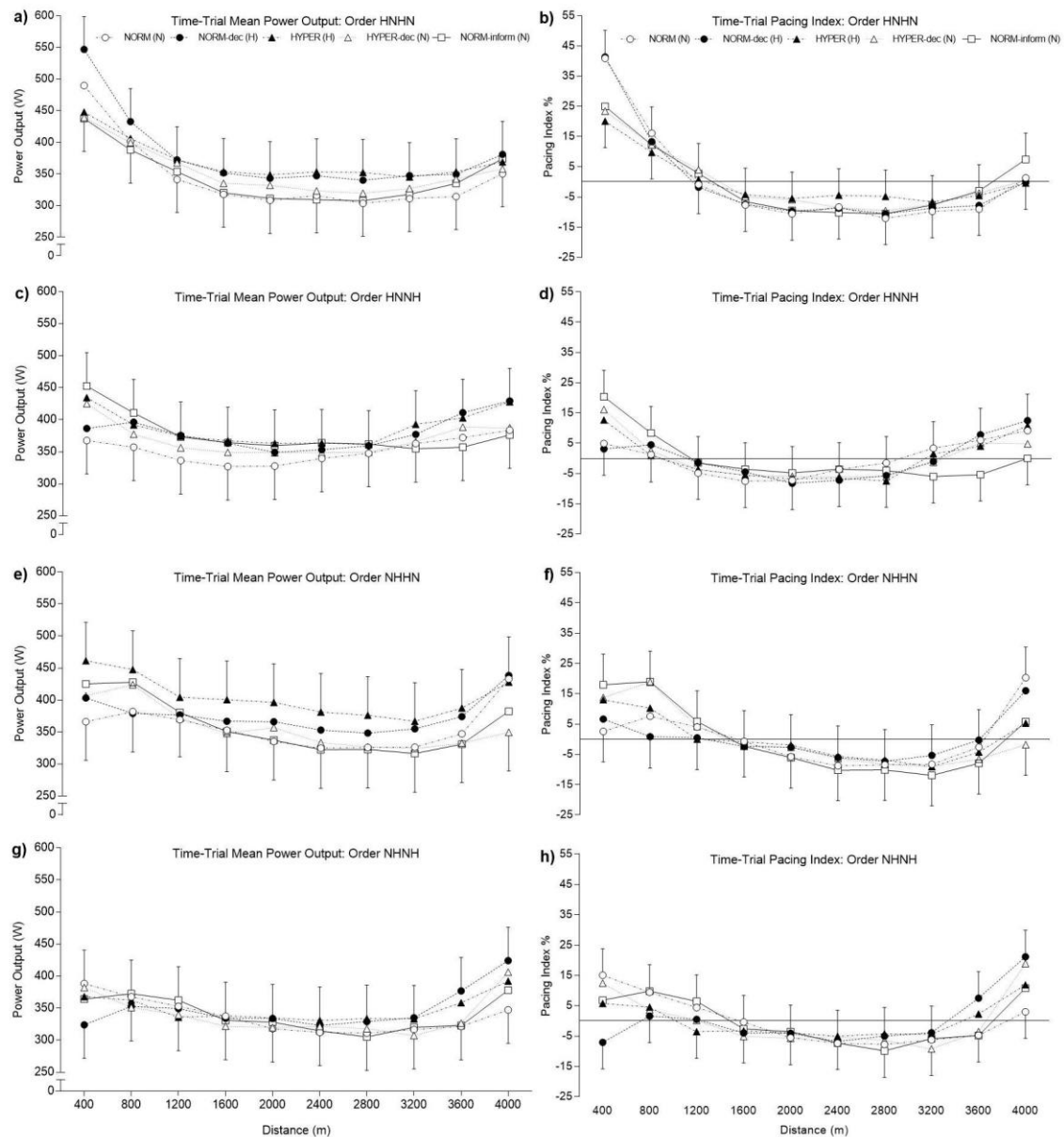


Figure 2. Mean power output (**left panel**) and associated pacing index (**right panel**) at 400-m intervals for each order group during 4000-m time-trials. (N) indicates normoxic inspired trials and (H) represents hyperoxic inspired trials. Results are reported as mean estimates \pm 95% confidence intervals.

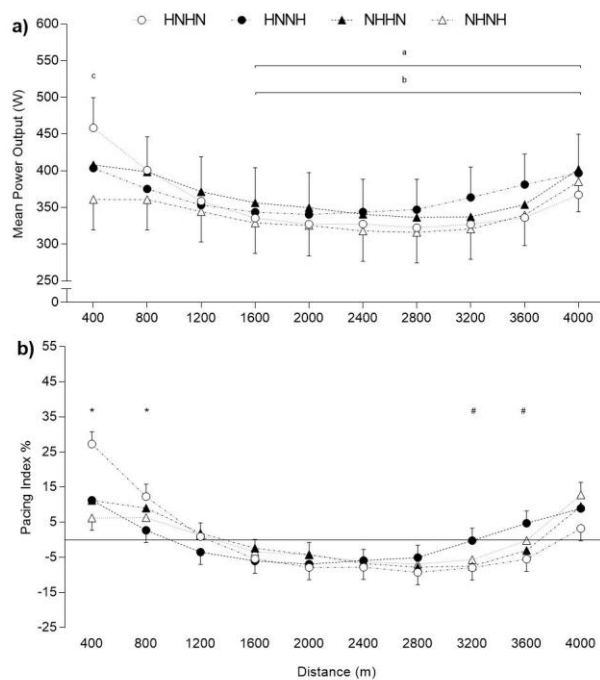


Figure 3. Mean power output for Trial Order by Segment interaction within the four trial orders (a) and associated pacing index (b) at 400-m intervals during 4000-m time-trials. (N) indicates normoxic inspired trials and (H) represents hyperoxic inspired trials. ^a Significant difference between HNHN and HNNH. ^b Significant difference between NHHN and NHHH. ^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 \pm 95% confidence intervals.

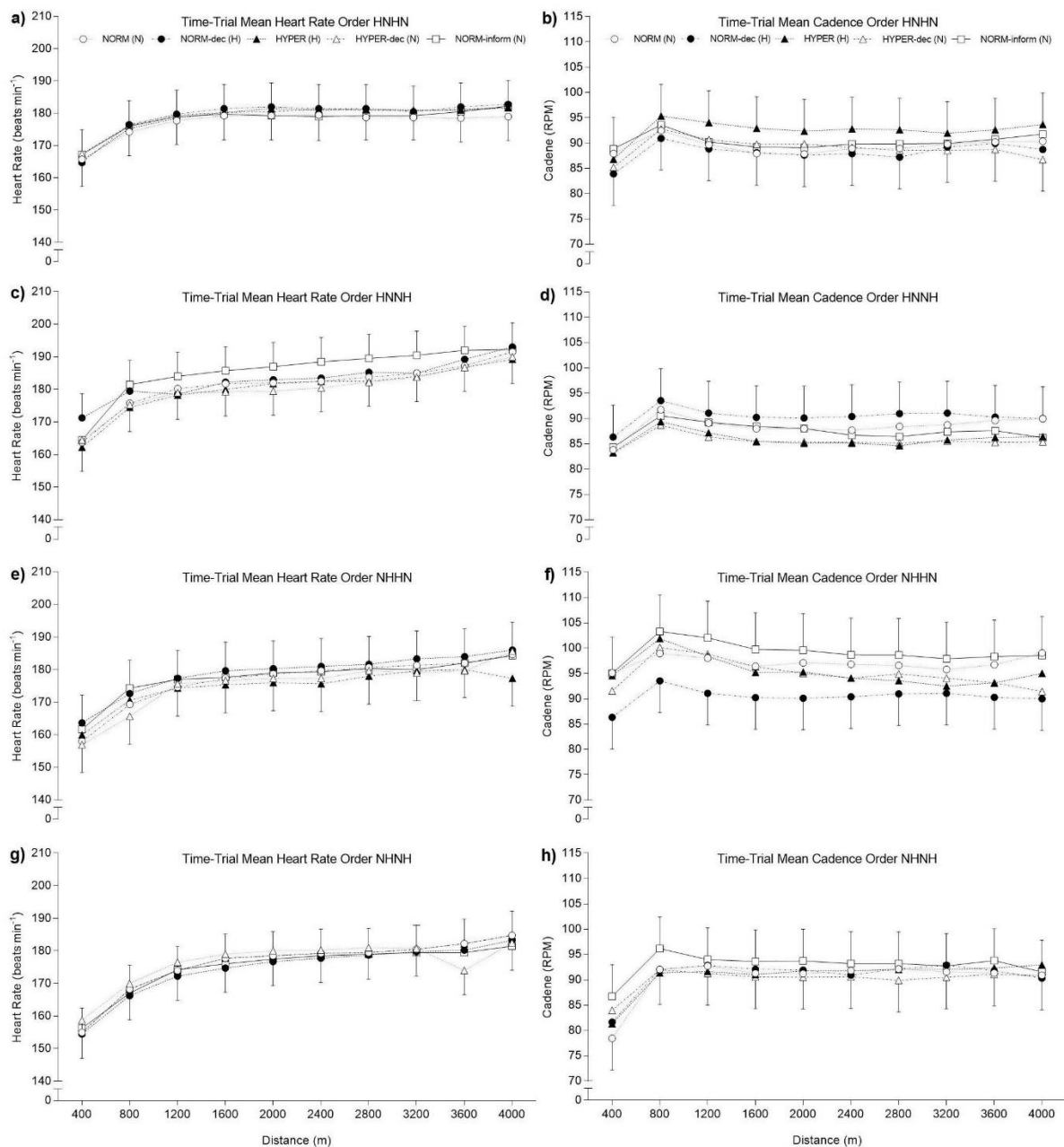


Figure 4. Mean heart rate responses (**left panel**) and cadence (**right panel**) at 400-m intervals for each order group during 4000-m time-trials. (N) indicates normoxic inspired trials and (H) represents hyperoxic inspired trials. Results are reported as mean estimates \pm 95% confidence intervals.

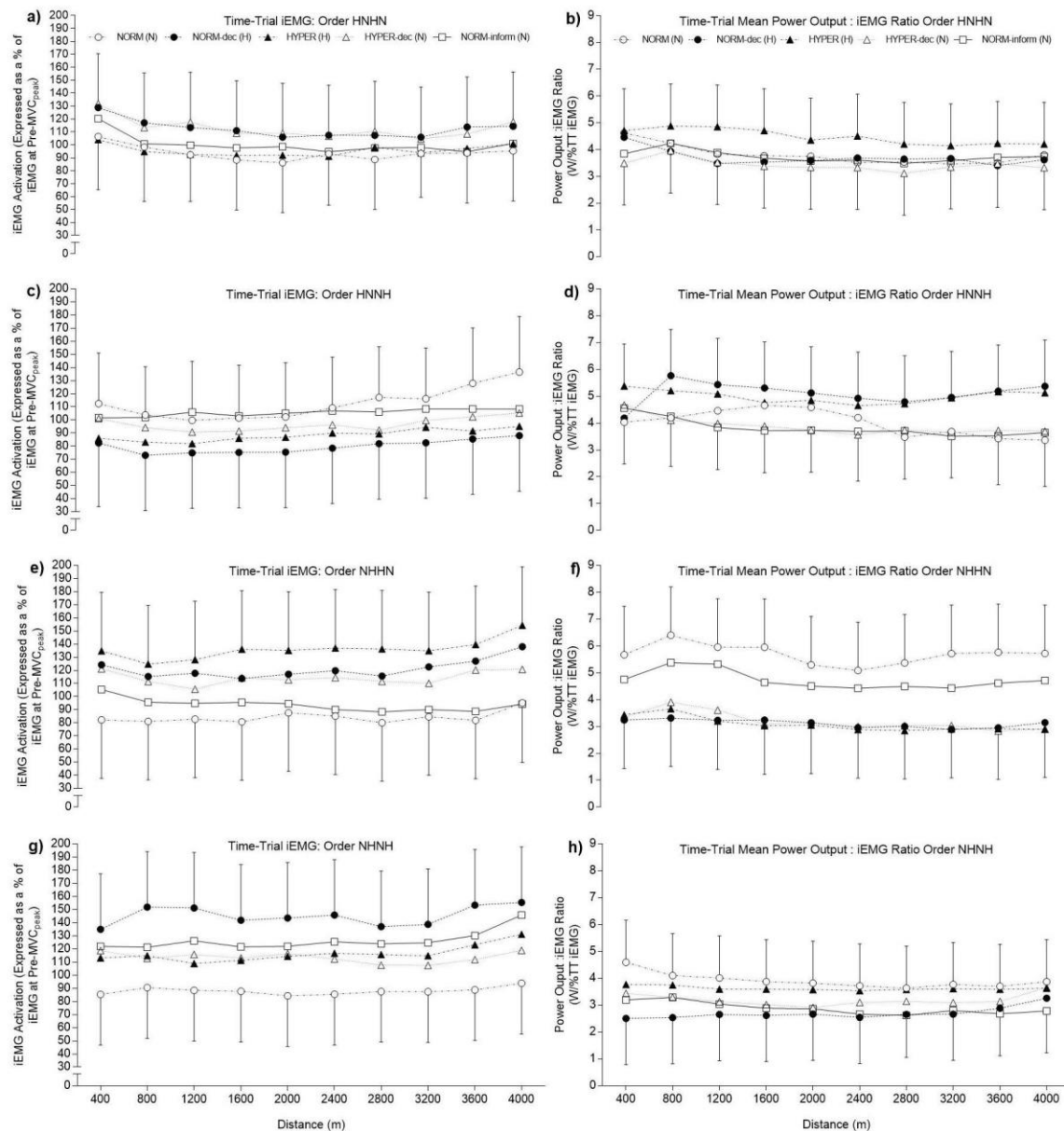


Figure 5. Mean integrated electromyography (iEMG) activity of the right vastus lateralis leg muscle (**left panel**), and mean ratio of power output to iEMG activity (**right panel**) at 400-m intervals for each order group during 4000-m time-trials. (N) indicates normoxic inspired trials and (H) represents hyperoxic inspired trials. Results are reported as mean estimates \pm 95% confidence intervals.

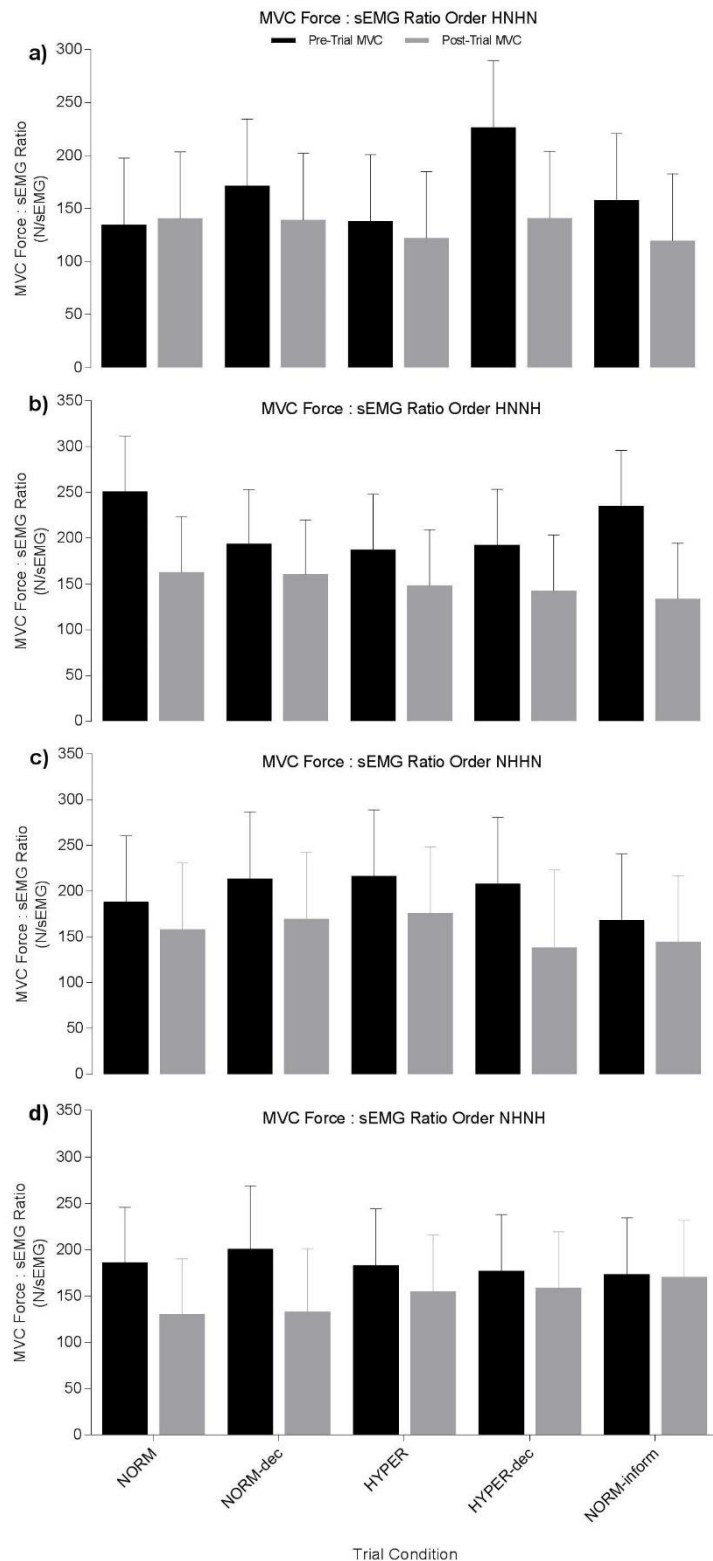


Figure 6. Ratio of peak force to surface electromyogram during pre- and post-trial maximal voluntary contractions. Results are reported as mean estimates \pm 95% confidence intervals.