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Deception improves TT performance in well-trained cyclists without augmented fatigue

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ABSTRACT

Purpose: To investigate the effects of feedback, in the form of a virtual avatar paced at 100 and 102% of baseline performance, on neuromuscular fatigue following a 4 km cycling time trial (TT). We hypothesised that improved cycling performance would occur due to participants exceeding a previously established critical threshold, and experiencing greater neuromuscular fatigue. **Methods:** Following familiarisation, ten well-trained cyclists performed a baseline 4 km TT without feedback (BASE), followed by two, 4 km TT where they raced against an avatar (set at 100% [accurate - ACC] and 102% [deception - DEC] of baseline power output), in a randomised and counterbalanced order. Before and after each TT, neuromuscular fatigue was assessed using maximal isometric voluntary contractions (MVC) of the quadriceps, and supramaximal electrical stimulation of the femoral nerve, during and 2 s after MVCs to assess voluntary activation and potentiated twitch force. Blood lactate was taken pre- and post-trials and rating of perceived exertion (RPE) was taken throughout each TT. **Results**: TT performance improved following deception of feedback compared to baseline performance (−5.8 s, P = 0.019). Blood lactate increased following DEC compared to BASE (+1.37 mmol·L^{-1}, P = 0.019). Despite this, there was no difference in any measures of exercise-induced neuromuscular fatigue (P > 0.05). Similarly, RPE was not different between trials. **Conclusion:** Well-trained male cyclists can improve cycling TT performance when competing against an avatar increased to 102% of a previously established best effort. However, this improvement is not associated with a measurable augmentation of neuromuscular fatigue.

Words: 250

Key words: critical threshold, deception, fatigue, feedback.

INTRODUCTION

The pacing strategy adopted during a cycling time trial (TT) is intended to optimize performance while minimizing fatigue (1, 2). Fatigue is a universal phenomenon characterized by sensations of tiredness and weakness during or following exertion, which is underpinned and/or modulated by multiple physiological and psychological processes. An acute bout of exercise, and the consequent disruption to homeostasis, is a particularly potent stimulus to elicit fatigue. The potential contributors to the fatigue experienced during cycling TT exercise include energy depletion (3), cardiorespiratory stress (4), disruption to peripheral homeostasis (5), reduced muscle activation (6), and muscle tissue damage (7), all of which contribute to reductions in the force producing capacity of working muscles. For locomotor exercise a common approach to understanding the etiology of fatigue involves assessment of the neuromuscular adjustments underpinning the post-exercise reduction the in voluntary force producing capabilities of the involved muscles (8-11). Such investigations have demonstrated that high-intensity cycling exercise elicits central (i.e., an inability to voluntarily activate muscle) and peripheral (i.e., decrements in measures of contractile function) fatigue which contributes to this voluntary force loss, termed muscle fatigue (6). The relative magnitude of central and peripheral adjustments varies depending on the intensity and duration of the cycling (10, 11).

The contractile impairments observed after self-paced high-intensity cycling TTs are remarkably consistent between trials (12), with end-exercise quadriceps potentiated twitch force reductions of approximately 35% reported by numerous research groups (11-14). The magnitude of this reduction is consistent on repeated trials, and unaffected by pre-fatiguing exercise (15-17), or altered fractions of inspired oxygen (13). This phenomenon has been termed the 'critical threshold of peripheral fatigue' (12) and is believed to be a significant factor in high intensity exercise tolerance as participants cannot, or will not, voluntarily exceed this limit. However, such a critical threshold is specific to the task (12) and the unvarying degree of peripheral fatigue observed at the end of high-intensity self-paced cycling does not represent an absolute limit for peripheral fatigue. Rather, this supports the idea that exercisers maintain a "contractile reserve", perhaps as a protective mechanism in response to the threat exercise poses to the health of the organism (12, 18). Whether the exerciser can access this reserve under exceptional circumstances is debatable. Currently, the only evidence to suggest so are observations made when group III/IV afferent neurons are pharmaceutically blocked, enabling participants to willingly cycle past the point of peripheral fatigue attained when afferent feedback is intact (19, 20). This suggests a contractile reserve exists at task termination when afferent neurons

are intact, which if accessible under normal conditions, could conceptually allow for a greater performance.

One potential intervention that could motivate participants to tolerate a greater magnitude of peripheral fatigue, and thereby access a theoretical contractile reserve, is the provision of competition and/or surreptitious feedback. Previous literature investigating self-paced cycling has shown that performance is improved when receiving accurate feedback, or racing against a virtual avatar of a previous best performance (21, 22). Furthermore, Stone, Thomas (23) showed that when the speed of an avatar is surreptitiously increased by 2% (a "deception" trial), participants were able to improve cycling TT performance even further. Based on these findings, it could be suggested that the presence of an avatar increases motivation via an ego-orientated goal of beating the competition (24), which may enable athletes to tolerate a greater disruption to homeostasis, and the improved performance observed in these previous studies might have been associated with a higher-than-usual magnitude of neuromuscular fatigue.

Two recent investigations have provided partial support to this notion. Konings, Parkinson (25) showed that participants completing a 4 km cycling TT against a virtual avatar of a previous performance, improved performance and experienced greater losses in MVC, and potentiated twitch force (indicative of peripheral fatigue). This increased magnitude of peripheral fatigue occurred whilst rating of perceived exertion (RPE) did not change between trials. The authors attributed this to a shift of focus from internal to external factors, distracting participants from the discomfort elicited by the higher exercise intensity, and altering the decision making process associated with pacing. Additionally, Ducrocq, Hureau (26) demonstrated that when performance of a 5 km TT is improved by provision of surreptitious feedback (a 2% deception trial, similar to Stone, Thomas (23), (27)), participants experienced greater reductions in MVC, and increased central fatigue post-exercise, but no additional peripheral fatigue. During the deception trial greater motor unit recruitment of the quadriceps (increased electromyographic activity; rmsEMG) was also evident. These increases in rmsEMG have been previously suggested to have some association with the extent of anaerobic metabolism (28), which lends some support to the idea that deceiving participants could enable access to a previously protected metabolic reserve (23). Although reductions in voluntary force and voluntary activation were augmented with deceptive feedback, the involuntary twitch response to stimulation was not different between trials, indicating that a critical threshold of peripheral fatigue was not exceeded and a contractile reserve of the quadriceps was not utilized.

The population tested in Ducrocq, Hureau (26) were recreationally active, and their performance of 5 km TTs was modest (mean power output, 219 W). In contrast, well-trained cyclists would expect to attain a mean power output in excess of 330 W for the same TT (29). Similarly, despite Konings, Parkinson (25) stipulating that 'trained athletes' were tested, the mean power outputs reported for a 4 km TT (~280 W) would indicate this participant group were not well-trained cyclists (30). It has long been established that experienced, elite athletes employ different cognitive 'coping' strategies during endurance exercise to less experienced athletes (31), which might alter the motivational reaction to an avatar of either a previous best performance, or a deceptively increased performance. Therefore, previous findings related to the use of deceptive feedback and (25, 26) might not apply to well-trained endurance athletes, and it remains to be seen whether surreptitiously altered feedback improves performance by enabling the use of a contractile reserve in this population. Accordingly, the aim of the present study was to test the hypothesis that provision of surreptitious feedback during TT exercise in well-trained male cyclists would result in improved performance, and a concomitant greater magnitude of end-exercise peripheral fatigue.

METHODS

Participants

Ten well-trained cyclists (mean ± SD age, 29 ± 8 years; stature, 180 ± 6 cm; mass, 73 ± 8 kg; maximum aerobic power, 405 ± 27 W; 5.6 ± 0.5 $W \cdot kg^{-1}$; maximum oxygen uptake, 67.9 ± 6.8 $ml \cdot kg^{-1} \cdot min^{-1}$) volunteered and gave written informed consent for the study. Participants were informed that the study aimed to assess the reliability of physiological and neuromuscular responses to 4 km TTs, and were informed of the deception after completion of the study. Institutional ethical approval was granted, and the study adhered to the Declaration of Helsinki.

Experimental Design

Participants visited the laboratory on 4 separate occasions to complete a preliminary visit (ramp test and practice 4 km TT), followed by three experimental, self-paced 4 km cycling trials. The first self-paced trial was a 4 km TT to establish baseline performance. The final two visits were 4 km TT with either accurate or deceptive feedback, in a randomised and counterbalanced order. During all trials, participants were instructed to complete the distance as fast as possible. Each trial was scheduled for a similar time of day to account for diurnal variations in the cardiovascular and neuromuscular systems

(32, 33). Prior to each experimental trial participants were asked to refrain from ingesting caffeine (12 hours) and alcohol (24 hours), and performing strenuous exercise (24 hours).

Procedures

Preliminary visit

Participants completed a ramp test and 4 km TT on a Velotron Pro cycling ergometer (Velotron Racer Mate, Seattle, USA). The ramp test involved a 10 minute warm up at 100 W followed by a continuous incremental ramp in power of 1 W every 2 s (30 $W \cdot min^{-1}$) to the limit of tolerance. The test was terminated when cadence reduced by 20 rpm below participants' self-selected cadence. Expired air was analysed via an online breath by breath system (Oxycon Pro, Care Fusion, Hoechberg, Germany). Ventilatory volumes were inferred from the measurement of gas flow using a digital turbine transducer (volume, 0–10 L; resolution, 3 mL; flow, 0–15 $L \cdot s^{-1}$) attached to a mask. Maximum oxygen uptake (VO_2max) was calculated as the highest 30 s mean value attained before test termination, and the end test power was recorded as maximum aerobic power (P_{max}). A practice 4 km TT was included in the preliminary visit to familiarise participants with the exercise and thus limit learning effects in the experimental trials (29).

Experimental Visits

Participants completed three, 4 km TT. Before and immediately after each trial, a neuromuscular function assessment was completed (see below). All trials were completed following a standardised warm up (5 min at 150 W then 5 min at 70% P_{max} followed by 5 min of rest). Ratings of perceived exertion (RPE) were obtained every km. Blood lactate was measured using 20 μL capillary blood samples taken from the fingertip 2 min prior to the start, and immediately post-trial (Biosen, EKF Diagnostic, Barleben, Germany). The first trial (baseline; BASE) was performed with participants shown their progress in real time on a screen via an avatar and a graphic showing distance covered. All other feedback was removed from the screen. The same flat course profile was used in all subsequent trials. The next two trials were performed with two avatars on screen; one showing their current performance, and a pacemaker avatar showing their baseline performance (accurate; ACC) or the baseline power output increased by 2% (deception; DEC). The 2% margin of increase was specifically chosen as it is the smallest worthwhile change in 4 km performance (29), thus providing the least chance of being detected by the participant. Provision of augmented feedback equating to a 2% increase in power output has previously been successfully used to elicit performance improvements in well-trained cyclists performing similar TTs in our laboratory (23, 27). It was

confirmed that participants believed they were racing their baseline performance in both experimental trials and none suspected the deception at any point throughout the study.

Neuromuscular Function

Neuromuscular function was assessed before and immediately after each TT. This consisted of 3 maximal isometric knee-extensor contractions (MVC) separated by 30 s rest, with femoral nerve stimulation delivered at peak force and 2 s following each MVC to calculate voluntary activation (VA) and measure the potentiated quadriceps twitch force ($Q_{tw,pot}$). Following task termination, each 'post-trial' neuromuscular function assessment was completed in <1.5 min.

Force and EMG Recording

During the neuromuscular function assessments, participants sat upright in a custom built chair with hips and knees at 90˚ flexion. A calibrated load cell (MuscleLab force sensor 300, Ergotest Technology, Norway) was attached via a noncompliant cuff positioned on the participant's right leg, superior to the malleoli, to measure knee extensor force (N). Surface Ag/AgCl electrodes (Kendall H87PG/F; Covidien, Mansfield, MA) were placed 2 cm apart over the *vastus lateralis* (VL) to record the compound muscle action potential (M-wave), elicited by the electrical stimulation of the femoral nerve. Skin was shaved and abraded to ensure minimal impedance, then electrodes were positioned according to the SENIAM guidelines, a reference electrode was placed over the patella. Electrode placement was marked with permanent ink to ensure a consistent placement between trials. Signals were amplified: (gain ×1000 for EMG and ×300 for force; CED 1902, Cambridge Electronic Design, Cambridge, UK), bandpass filtered (EMG only: 20–2000 Hz), digitized (4 kHz; CED 1401, Cambridge Electronic Design), and analysed offline (Spike2 v7.12, Cambridge Electronic Design).

Motor Nerve Stimulation

Single electrical stimuli (200 μs duration) were applied to the right femoral nerve using a constant-current stimulator (DS7AH Digitimer Ltd., Welwyn Garden City, UK) via adhesive surface electrodes (CF3200; Nidd Valley Medical Ltd., Harrogate, UK) at rest, and during voluntary contractions. The cathode was positioned over the nerve high in the femoral triangle, in the location that elicited the maximum quadriceps twitch amplitude (Q_{tw}) and the M-wave (M_{max}) at rest. The anode was positioned midway between the greater trochanter and the iliac crest. The optimal stimulation intensity was determined as the minimum current that elicited maximum values of Q_{tw} and M_{max} at rest. To ensure a supramaximal stimulus, and to account for fatigue-dependent changes in axonal excitability, the

intensity was increased by 30% and was not different between trials (171 ± 34, 167 ± 38, and 164 ± 29 mA, P = 0.722).

Data Analysis

VA measured via motor nerve stimulation was quantified using the twitch interpolation method: VA (%) = (1 - [SIT ÷ $Q_{tw,pot}$] × 100), where SIT is the mean amplitude of the superimposed twitch force measured during MVCs, and $Q_{tw,pot}$ is the mean amplitude of the resting quadriceps potentiated twitch force assessed 2 s post-MVC (34). The peak-to-peak amplitude and the area of the evoked M_{max} responses were quantified offline. Cycling power output (W) was recorded during each TT and was averaged over 10% distance epochs. Between day reliability values (coefficient of variation – CV%) for MVC (4.3%), $Q_{tw.pot}$ (6.9%), and VA (1.7%) were calculated *post hoc* using the 'pre' data of the three experimental trials.

Statistical Analysis

Two-way (trial × time) repeated measures ANOVAs were used to assess within and between-trial differences in neuromuscular measures (MVC, $Q_{tw,pot}$, M_{max} amplitude and area, within twitch characteristics), and cycling performance (power). A one-way repeated measures ANOVA was conducted to assess between trial differences in time taken to complete the TT, and pre-post changes in blood lactate. For all parametric ANOVAs, Bonferroni pairwise comparison tests were run *post-hoc* when a significant main effect was observed. A Friedmann's ANOVA with *post hoc* Wilcoxon signed-ranks test was used for nonparametric data (RPE). The assumptions underpinning these statistical procedures were verified, and all data were considered normal. Descriptive data are presented as means ± SD in text, tables, and figures. Statistical significance was assumed at $P \leq 0.05$.

RESULTS

Participant Characteristics

Maximum aerobic power achieved during the initial ramp test was 5.3 ± 0.7 W·kg^{-1}. According to De Pauw et al. (2013), the values placed 1 participant in the 'trained' category (4.6 W·kg^{-1}), 7 participants in the 'well-trained' category (range: 4.6 – 5.5 W·kg^{-1}), and 2 participants in the 'professional' category (range: 6.1 – 6.8 W·kg^{-1}).

4 km Time Trial Performance

Time taken to complete 4 km TT was different between trials (BASE, 367 ± 15 s; ACC, 365 ± 18 s; DEC, 361 ± 17 s; $F_{2,20}$ = 5.40; P = 0.015; η^2 = 0.375). Nine out of ten participants improved their 4 km TT during DEC compared to BASE (range: –2 to –15 s), with one recording an equal time. Eight participants were faster in DEC compared to ACC (range: –2 to –15 s), and two participants were not (range: 0 to +6 s). Pairwise comparisons revealed that DEC was faster than BASE (−5.80 ± 1.65 s; P = 0.019), however ACC was not (−1.70 ± 1.84 s; P = 1.000). There was no significant difference between DEC and ACC (−4.10 ± 1.94 s, P = 0.191; Table 1). Mean power profiles can be seen in Figure 1, the ANOVA revealed no significant effect between trials (BASE, 324 ± 38 W; ACC, 327 ± 42 W; DEC 334, ± 41 W; $F_{2,20}$ = 0.569; P = 0.576; η^2 = 0.059). And no interaction effect was shown (trial × time: $F_{18,180}$ = 1.31; P = 0.186; η^2 = 0.127). The increase in blood lactate (Table 1) was significantly different between trials (trial effect: $F_{2,20}$ = 4.69; P = 0.014; η^2 = 0.378). Pairwise comparisons revealed that blood lactate increased more in DEC than BASE (Δ13.71 vs. Δ12.34 mmol.L^{-1}; P = 0.019), however, there was no difference between ACC and DEC or BASE (Δ13.09 vs Δ12.34mmol.L^{-1}; P = 0.161). Mean RPE did not differ between trials (X^2_2 = 1.04; P = 0.595).

TABLE 1 HERE

FIGURE 1 HERE

Neuromuscular Function

MVC force decreased in all trials (BASE: -21 ± 6%; ACC: -19 ± 6%; DEC -23 ± 7%; $F_{2,20}$ = 226.40; P < 0.001; η^2 = 0.962). However, there was no trial ($F_{2,20}$ = 0.45; P = 0.646; η^2 = 0.0471) or trial × time interaction effect ($F_{2,20}$ = 0.82; P = 0.456; η^2 = 0.083, Figure 2, panel A). Voluntary activation showed the same pattern, decreasing from pre- to post-exercise (BASE: -14 ± 11%; ACC: -12 ± 9%; DEC -13 ± 12%; $F_{2,20}$ =18.61; P = 0.002; η^2 = 0.674) with no difference in the response between trials (trial: $F_{2,20}$ = 0.70; P = 0.511; η^2 = 0.072 and interaction effect: $F_{2,20}$ = 0.191; P = 0.828 η^2 = 0.021, Figure 2, panel B). Potentiated twitch force also declined in each trial (BASE: -34 ± 17%; ACC: -35 ± 12; DEC -41 ± 14%; time effect: $F_{2,20}$ = 54.90; P < 0.001; η^2 = 0.859), and similar to MVC and VA, there was no difference between trials (trial: $F_{2,20}$ = 1.16; P = 0.337; η^2 = 0.114, trial × time interaction: $F_{2,20}$ = 1.25; P = 0.311; η^2 = 0.122, Figure 2, panel C). Evoked EMG variables (i.e., M_{max} amplitude and area, Table 2) were not different between trials and did not change from pre- to post-trial (P > 0.05). Within twitch characteristics (Table 2) all decreased from pre- to post-trials (P < 0.05), however the degree of change was not different between trials (P > 0.05).

FIGURE 2 HERE

TABLE 2 HERE

DISCUSSION

The aim of the present study was to investigate whether improvements in 4 km TT performance elicited by deceptive feedback in well-trained cyclists, was associated with increased end-exercise peripheral fatigue. The present study shows that cycling performance was improved by competition against a virtual avatar surreptitiously changed to 102% of a previous best effort, but not against an avatar set at 100%. This improvement was accompanied by an increase in blood lactate at task completion and supports previous literature utilising this method to improve performance (23, 27). Despite the improvement in TT completion and increase in blood lactate in the deception trial, there was no augmentation of peripheral fatigue. Consequently, the hypothesis that performance improvements elicited by deceptive feedback can be explained by exceeding or altering a previously established critical threshold of peripheral fatigue is not supported by the present study.

In contrast to our findings, Ducrocq, Hureau (26) and Kronings et al. (2017) reported greater decreases in neuromuscular function when competing against a virtual avatar and receiving deceptive feedback. Ducrocq, Hureau (26) showed a 2% improvement in completion time following the 102% pacemaker trial compared to the accurate feedback trial, with measures of maximum voluntary force decreasing an extra 5% (−41 vs. −36%) and voluntary activation an extra 4% (−18 vs. −14%) post-exercise. The authors suggested that the increased power output, cardiovascular response, and muscle activation during the deceptive trial led to increased metabolic work and consequently an accumulation of deleterious metabolites within the quadriceps. Despite this, and in agreement with the present study, the improved performance in the deceptive trial was not associated with any additional peripheral fatigue (reductions in potentiated twitch force); a measure of muscle function that does not rely on voluntary effort. This would suggest that a critical threshold for peripheral fatigue was not exceeded and a contractile reserve was not utilised. The differences observed in MVC and VA, which rely on voluntary maximal efforts, could suggest that central fatigue was greater following the deception trial. However, this was not a limiting factor to performance as participants significantly improved compared to a baseline trial without feedback (26). It is possible that the recreationally active participants in Ducrocq, Hureau (26) were consciously aware of the exaggerated fatigue in the deception trial, as evidenced by an increase in RPE, and consequently any reductions in voluntary

force could be attributed to an increased perception of effort rather than an exaggerated disruption to peripheral homeostasis.

Similar to the present study and others (27, 35), Konings, Parkinson (25) reported no differences in RPE when competing against an avatar of previous performance. They did however, find that potentiated doublet twitches were significantly smaller following a competitive trial, indicating that the improved performance was concurrent with additional peripheral fatigue. In contrast, well-trained cyclists in the present study did not improve performance or experience a greater degree of peripheral fatigue following accurate feedback, only improving performance following deceptive feedback. This might suggest that well-trained cyclists are able to tolerate an increased task demand without compromising their voluntary force capacity, and the reduced voluntary measures of neuromuscular fatigue observed by Ducrocq, Hureau (26) could be a consequence of a less-than-maximal effort in an unaccustomed population, rather than a higher degree of neuromuscular fatigue *per se*. This can be seen by the ~30-40% decrease in MVC in the recreationally active cohort (26), compared to 22% in well-trained cyclists in the present study. The differences in end-exercise peripheral fatigue between the present study and Konings, Parkinson (25) might lie in the differences in training status. In the present study participants completed the 4 km TT in ~365 s, with a mean power output of ~325 W, compared to ~384 s and ~280 W in Konings, Parkinson (25).

The lack of difference shown in post-exercise neuromuscular function in the present study, despite the improved performance, could be explained by the small differences observed between trials. It has previously been shown that the post-exercise degree of central and peripheral fatigue varies with task intensity and duration (10, 11). Whilst this would suggest that a difference might be seen, given that cyclists in the present study improved their performance, it is plausible that the small 10 W (3%) improvement in mean power output simply was not of a large enough magnitude to elicit a more substantial amount of neuromuscular fatigue. It could also be suggested that the efficacy of deception shown in the present and previous data, might not apply to longer duration time trials as the limiting factors to performance are different (11). An alternative explanation for the lack of difference could be that the methods used in the present study were not sensitive enough to detect any difference. Between-day reliability values (CV%) from the present study, along with previous work from our laboratories, indicate good or excellent reliability for outcome measures under study; MVC, 4.1 – 5.9%; $Q_{tw.pot}$, 4.8 – 6.6%; and VA, 1.7 – 3.1% (10, 36). Thus, the differences shown by Ducrocq et al.

(2017) (MVC, +5%, VA, +4%) are on the limit of measurement error, and might not have been detectable in the present study.

Another potential explanation for the lack of difference between trials could relate to the altered pacing strategy in the deception trial, which might have improved performance by ensuring a more optimal distribution of work, in a manner that would not necessarily lead to augmented neuromuscular fatigue. The lack of trial effect for mean power output showed that well-trained participants did not simply cycle faster for the entire time trial to beat the deceptively paced avatar. Alternatively, they might have altered their pacing strategy in order to perform a similar amount of work, but still beat the avatar. Recent evidence suggests that faster pulmonary oxygen kinetics during the initial stages of 6 mins of constant-load cycling leads to reduced peripheral fatigue at task termination (37). The present study utilised self-paced cycling of similar duration (~6 mins). Therefore, despite the non-significant interaction effect in power output, it might have been the case that increased power output in the initial stages of DEC (0 - 1200 m: mean difference 25 W, 8 out of 10 participants) caused participants to reach peak aerobic energy production faster, reducing the reliance on anaerobic respiration, negating the need for a larger 'end spurt' to beat the avatar. This would negate the potentially deleterious effects one would expect from increased power output, paradoxically leading to no difference in post-exercise neuromuscular fatigue between trials, despite an improved performance.

The present data suggest that a deceptively paced avatar is an effective way to improve 4 km TT performance in well-trained cyclists. However, unlike lesser-trained populations (25, 26), the mechanisms of performance enhancement are not due to augmented neuromuscular output and consequent fatigue. More likely, well-trained athletes alter the pacing strategy employed to beat an avatar of their previous best performance. Therefore, the use of surreptitiously altered avatar may not be an effective training method when intending to increase the metabolic and neuromuscular stress imposed on the athlete for a given task.

One limitation of the present data was sample size, albeit 10 participants is comparable to the sample size of Ducrocq et al. (26): n=11 and Konings et al. (25): n=12. However, it may be the case that the ANOVA used to compare differences between trials was too robust to show differences. A post hoc power calculation was conducted using the trial*time interaction effect size (η^2 = 0.122). The present data has a statistical power of 0.66, and a sample size of 13 would have achieved a statistical power

of 0.8. Additionally, the difference in $Q_{tw,pot}$ reduction between BASE and DEC, appeared large enough (~7%) to reflect a meaningful change. However, when the individual changes in $Q_{tw,pot}$ reduction are plotted across trials, no clear trend was observed (data not shown). Furthermore, there was no correlation between differences in time to completion and $Q_{tw,pot}$ reduction ($r^2 = 0.13$, $P = 0.314$).

CONCLUSION

The results of the present study suggest that improved performance via deception does not cause a critical threshold of peripheral fatigue to be exceeded in well-trained participants. More likely, the improved performance is due to alterations in pacing strategy. Future research should look to investigate the mechanisms underpinning performance enhancements via deceptive feedback and competition in athletes and non-athletes, as this remains unclear based on the current evidence.

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Conflicts of Interest

The authors report no personal, financial or other conflicts of interest are declared by the authors. The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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Table and Figure Legends

Table 1. Performance, rating of perceived exertion, and blood lactate responses to 4 km time-trials at baseline and with accurate and deceptive feedback. * $P < 0.05$ significantly different from pre-.

Table 2. Measures of evoked EMG and within twitch variables pre and immediately post (< 1.5 min) 4 km time-trials at baseline and with accurate and deceptive feedback. * $P < 0.05$ significantly different from pre.

Figure 1. Power output during the 4 km time-trials displayed over 10% epochs. # = $P < 0.05$ significant interaction effect, * $P < 0.05$ deception significantly different from baseline.

Figure 2. Changes in neuromuscular function from pre to post each time-trial. MVC, maximal voluntary contraction (A); VA, voluntary activation (B) and $Q_{tw,pot}$, quadriceps potentiated twitch force (C). For each variable, individual data are shown as the unfilled symbols and the group mean is shown as the filled symbols.

Table 1

Table 1. Performance, perceptual and haematological measures pre, during and post the 4 km time-trials at baseline and with accurate and deceptive feedback.

	Baseline	Accurate	Deception
Time to completion (s)	367 ± 15	365 ± 18	361 ± 17[#]
Mean power output (W)	324 ± 38	327 ± 42	334 ± 41
% maximum power	80 ± 7	81 ± 8	82 ± 7
Mean RPE	8 ± 1	8 ± 1	8 ± 1
Blood Lactate ($mmol \cdot L^{-1}$)			
Pre	1.88 ± 0.50	2.02 ± 0.77	1.71 ± 0.46
Post	14.21 ± 2.49*	15.11 ± 1.72*	15.41 ± 1.82*[#]

Values are means ± SD for 10 participants. RPE = rating of perceived exertion. * $P < 0.001$ vs. Pre.
[#] $P < 0.05$ vs. Baseline

Table 2

Table 2. Measures of evoked EMG and within twitch variables pre and immediately post (< 1.5 min) 4 km time-trials at baseline and with accurate and deceptive feedback.

			Baseline	Accurate	Deception
M_{max}	**Amplitude**	**Pre**	9.1 ± 4.8	8.2 ± 3.6	9.2 ± 4.0
	(mV)	**Post**	9.5 ± 5.7	8.1 ± 3.6	8.7 ± 4.2
	Area	**Pre**	57.5 ± 29.6	56.5 ± 25.9	70.1 ± 36.1
	(mV·s^{-1})	**Post**	71.5 ± 54.5	60.9 ± 26.8	70.0 ± 38.6
Within twitch Characteristics	**MRFD**	**Pre**	5001 ± 1214	4954 ± 1066	5552 ± 1472
	(N·s^{-1})	**Post**	2865 ± 720*	3105 ± 858*	2853 ± 843*
	MRR	**Pre**	−1398 ± 402	−1921 ± 543	−1628 ± 551
	(N·s^{-1})	**Post**	−1041 ± 429*	−1354 ± 442*	−1183 ± 643*
	CT	**Pre**	84 ± 10	84 ± 9	78 ± 11
	(ms)	**Post**	68 ± 6*	70 ± 7*	68 ± 4*
	RT0.5	**Pre**	82 ± 19	83 ± 10	81 ± 12
	(ms)	**Post**	71 ± 19*	67 ± 14*	78 ± 17*

Values are means ± SD for 10 participants. M_{max}, maximal M-wave; MRFD, maximum rate of force development; CT, contraction time; MRR, maximum relaxation rate; RT0.5, one-half relaxation time. * P <0.05 different from pre.

Figure 1

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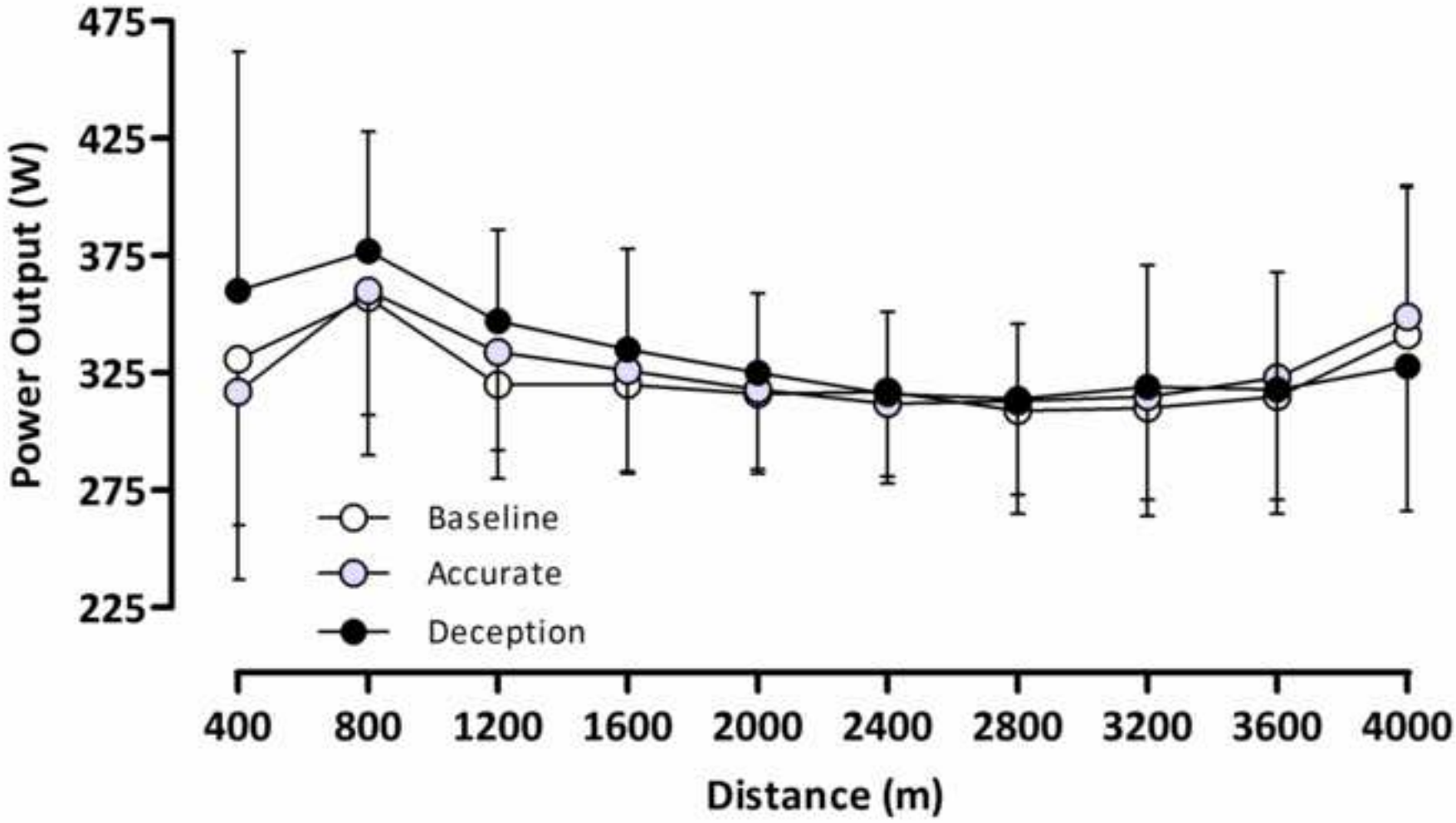

Figure 2

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