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Increased fatigue response to augmented deceptive feedback during cycling time-trial

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Guillaume P. Ducrocq, Thomas J. Hureau, Olivier Meste and Grégory M. Blain. *Increased fatigue response to augmented deceptive feedback during cycling time-trial.*

Purpose: This study aimed to investigate the effect of different magnitudes of deception on performance and exercise-induced fatigue during cycling time-trial.

Methods: Following three familiarization visits, three females and eight males performed three 5 km cycling time-trials while following a simulated dynamic avatar reproducing either 100% (5K_{100%}), 102% (5K_{102%}) or 105% (5K_{105%}) of the subject's previous fastest trial. Quadriceps muscle activation was quantified with surface electromyography. Fatigue was quantified by pre- to post-exercise (10 s through 15 min recovery) changes in quadriceps maximal voluntary contraction (MVC) force, potentiated twitch force evoked by electrical femoral nerve stimulation (QT_{Single}) and voluntary activation (VA, twitch interpolation technique).

Results: Greater quadriceps muscle activation in 5K_{102%} vs. 5K_{100%} (12±11%) was found in parallel with a 5±2% and 2±1% improvement in power output and completion time, respectively ($p < 0.01$). Exercise-induced reduction in MVC force and VA were 14±19% and 28±31% greater at exercise termination (at 10 s) whereas QT_{Single} recovery (from 10 s to 15 min) was 5±5% less in 5K_{102%} vs. 5K_{100%} ($p < 0.01$). No difference in performance or fatigue indices measured at exercise termination was found between 5K_{100%} and 5K_{105%}.

Conclusion: Muscle activation and performance improvements during a deceptive cycling time-trial were achieved only with a 2% magnitude of deception and were associated with a further impairment in MVC force, QT_{Single} recovery and VA compared to control. Performance improvement during cycling time-trial with augmented deceptive feedback therefore resulted in exacerbated exercise-induced peripheral and central fatigue.

Key words: *Deceptive cycling time-trial; performance, muscle activation; peripheral fatigue; central fatigue*

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INTRODUCTION

During strenuous exercise, an athlete's performance is critically dependent upon the force- or power-generating capacity of the contracting muscles, which progressively declines until exercise termination. This transient reduction in the muscle ability to produce force or power defines neuromuscular fatigue (14). The mechanisms underpinning exercise-induced fatigue involve the alteration of processes within the contracting muscles (i.e. 'peripheral fatigue') and/or within the central nervous system, including the brain (i.e. 'central fatigue'). Specifically, intramuscular metabolic

perturbation determines peripheral fatigue by compromising the excitation – contraction coupling in muscle fibers (1). Peripheral fatigue is commonly quantified by the pre- to post-exercise reduction in twitch force evoked by electrical motor nerve stimulation. Central fatigue corresponds to a failure (or compromised willingness) of the central nervous system to activate the working muscles (14). Central fatigue can be estimated using twitch interpolation by comparing the pre- to post-exercise proportion of muscle force recruited voluntarily [i.e. voluntary activation (VA)] (23).

Growing evidence supports the concept that the termination of strenuous exercise coincides with a severe and task-specific degree of peripheral fatigue (3, 4, 8, 13, 17, 18, 30). As this level of fatigue is not typically exceeded by the exercising human with functionally intact muscle afferent feedback, it has previously been referred to as the 'critical' threshold of peripheral fatigue (4). This 'critical' threshold of peripheral fatigue, which is thought to protect the muscle against an abnormal homeostatic challenge (6), does not represent the muscles' ultimate contractile limit (2, 5, 6, 12, 21, 28). For example, during sustained isometric submaximal contraction of the triceps surae (21) or the knee extensors (28), participants reached task failure before any failure of the muscle excitation-contraction coupling. Indeed, when the muscle was stimulated electrically once the exercise could not be continued voluntarily, the targeted force was restored and peripheral fatigue further increased beyond the level measured at voluntary task failure (21, 28). These observations suggest that exercise is regulated to retain a contractile 'reserve' even at task failure (2). By using deceptively augmented feedback, athletes may be able to accumulate peripheral fatigue beyond the 'critical' threshold and tap into this contractile 'reserve' to improve their performance.

Various forms of deception have been shown to be effective in improving performance. For example, time to task failure during constant workload cycling exercise has been shown to be significantly longer with deceptive feedback using a slow running clock (27). It was speculated that the participants increased their effort and performance to offset their 'perceived' performance. Similarly, a shorter time to completion and greater power output were achieved during a cycling time-trial by misleading participants into believing they were competing against a virtual avatar reproducing the real-time performance of an opponent with similar ability to theirs when, in fact, it reproduced the participant's

previous best performance (11, 38). These performance improvements with deception were attributable to a greater anaerobic contribution to total energy expenditure and were associated with a reduced perceived exertion and attentional focus towards internal sensory feedback (e.g. leg fatigue, pain, heart beating or breathing frequency and depth) (11, 33, 37, 38). The combined increase in power output and anaerobic contribution to total energy expenditure might be linked to a higher muscle activation (i.e. recruitment of additional motor units and/or increase in motor units firing rate) and might result in a greater accumulation of intramuscular metabolic by-products known to determine peripheral fatigue (1, 6). However, to date, the neuromuscular consequences underpinning the performance improvement with deception are unknown. Specifically, it remains to be determined whether the improved power output and metabolic demand with an augmented deceptive feedback are associated with a significant increase in muscle activation and are dependent upon the access of the muscle contractile “reserve” (i.e. greater exercise-induced peripheral fatigue).

Moreover, most experimental approaches using deceptive feedback were based on a competitive simulation, during which participants were instructed to race against a virtual opponent (11, 33, 37, 38). These approaches may limit the application of deceptive feedback to training and may not be fully representative of competition at the highest level, as performance in elite cyclists or endurance runners rely significantly on pacemakers. In contrast, a deceptive approach, during which participants would be instructed to follow a virtual pacemaker (*vs.* race against a virtual opponent), would allow for accurate adjustments of the magnitude of deception and therefore optimize the performance improvement associated with augmented deceptive feedback. This novel approach may thus increase applicability and practicality of the deception strategy during training and competition.

Thus, using a virtual pacemaker, this study primarily aimed to determine the effect of different magnitudes of deception on exercise performance and exercise-induced fatigue during whole body endurance cycling exercise. A second objective was to characterize the metabolic, cardio-ventilatory and psychological responses to non-competitive deceptive cycling exercise. To this end, participants performed a 5 km cycling time-trial following a simulated dynamic avatar reproducing 100 %, 102 % and 105 % of the subject’s previous fastest trial. We hypothesized that, compared to control, improvements in power output and completion time during deceptive time-trial are associated with: 1) a further reduction in the participants’ potentiated twitch and maximal voluntary contraction forces, 2) a significant increase in muscle activation and metabolic work during exercise, 3) a significant increase in the cardio-ventilatory response to exercise to compensate for the increased metabolic work, and 4) a significant

reduction in perceived exertion and internal attentional focus during exercise..

METHOD

Subjects

Three females [Age (mean \pm SD): 22 \pm 2 years, height: 159 \pm 10 cm, body mass: 57 \pm 4 kg, body fat: 24 \pm 3 %] and eight males [Age: 21 \pm 2 years, height: 178 \pm 6 cm, body mass: 74 \pm 8 kg, body fat: 12 \pm 2 %] participated in this study. The number of subjects was determined a priori by statistical power analysis (G*Power, 3.1.9.2) using the following criteria: α error, 0.05; power level (1 – β), 0.85; effect size, 0.15; correlation among measure, 0.95. All participants were healthy, recreationally active, non-smokers and non-medicated. They were requested to prohibit physical activity for 48 h, alcohol and caffeinated beverage consumption for 24 h, and to have similar food intake (timing and content) prior to every experimental session. Written informed consent was obtained from each subject prior to the beginning of the study. All experimental procedures were approved by the local ethics committee and conducted according to the Declaration of Helsinki for human experimentation.

Experimental protocol

Participants visited the laboratory on eight to nine occasions. During preliminary visits, anthropometric measurements were collected and subjects were thoroughly familiarized with the neuromuscular and exercise testing procedures. During 3-4 practice sessions, all participants performed a 5km cycling time-trial on an electromagnetically braked cycle ergometer (Velotron, Elite Model, Racer Mate, Seattle, WA, USA), with freedom to alter power output by changing the gear ratio and/or pedalling frequency. Subjects remained seated throughout exercise. During each time-trial, an onscreen computer-generated avatar representing the participant’s progress as he/she undertook the time-trial and the distance covered during the time-trial were the only information displayed on a monitor placed directly in front of the participant. Participants were given strong vocal encouragement throughout exercise. Familiarization with the cycling time-trial modality was considered accomplished when the difference in power output between two successive practice trials was lower than 2 % (34). During the first experimental session, participants performed a control 5km time-trial without virtual pacer (5K_{CTRL}). The Velotron 3D software (RacerMate One) was used to store comprehensive performance data from 5K_{CTRL} and to replay the avatar riding at a similar or faster pace achieved during 5K_{CTRL} during future trials. At subsequent experimental visits, participants performed three deceptive 5km time-trials during which they were instructed to follow a second onscreen computer-generated avatar (i.e. a virtual pacemaker). Time-trials were therefore performed with a visual avatar representing current performance together with the virtual pacemaker, the distance covered and the

distance between the participant's avatar and the virtual pacemaker. Participants were asked to remain within 1-5m behind the virtual pacemaker. If they were not able to hold the pacemaker's pace, participants were asked to maintain the gap with the virtual pacemaker as small as possible and to complete the time-trial in the fastest time possible. During every session, participants were informed that the virtual pacemaker rode at a pace achieved during $5K_{CTRL}$. Although one of these trials accurately reproduced $5K_{CTRL}$ ($5K_{100\%}$), the virtual pacemaker's speed was set at 102% ($5K_{102\%}$) and 105% ($5K_{105\%}$) of $5K_{CTRL}$ in two other sessions (i.e. deceptive trials). In order to limit equivocal interpretations of any performance improvement with augmented deceptive feedback, the criteria for speed setting were based on an increase in power output at least two times greater than the coefficient of variation for power output measured during the last familiarization and $5K_{CTRL}$ time-trials (i.e. $1.7 \pm 0.9\%$). This setting also allowed us to test the influence of different magnitudes of deception on performance and neuromuscular fatigue. Finally, to determine the effect of an increase in the metabolic demand on peripheral fatigue during intense exercise and the progression of neuromuscular fatigue during the last kilometer of the time-trial, participants performed an additional $5K_{100\%}$ but their ride was stopped at 4 km ($4K_{100\%}$). Experimental sessions were performed on separate days and at the same time of day. To account for potential order effects, a randomization block design was used for the four experimental visits involving the virtual pacemaker.

Data collection and analysis

Contractile function and voluntary activation of the quadriceps.

For the assessment of the neuromuscular function, subjects were seated on a custom-made bench, arms folded across the chest, with a trunk/thigh angle of 135° and the right knee joint angle at 90° . A non-compliant strap attached to a calibrated load cell (model SM-2000N, Interface, Scottsdale, AZ, USA) was fixed to the subject's right ankle, just superior to the malleoli. The cathode, a self-adhesive electrode (3 x 3 cm, Ag-AgCl, Mini-KR, Contrôle-Graphique, Brie-Comte-Robert, France), was placed on the femoral triangle, at the stimulation site which resulted in both the maximal force output and the maximal amplitude of the compound muscle action potential (M_{MAX}) for the vastus lateralis (VL) and the vastus medialis (VM). The anode, a carbon-impregnated electrode (70 x 50mm) was rubbed with conductive gel and placed mid-way between the right antero-superior iliac spine and the great trochanter. The position of these electrodes was marked with indelible ink to ensure a reproducible stimulation site across visits. A constant current stimulator (DS7A, Digitimer, Hertfordshire, United-Kingdom) delivered a square wave stimulus (1 ms) at a maximum of 400V. To assure maximal spatial recruitment of motor units during these

tests, the stimulation intensity (69 ± 19 mA) was set to 120% of the stimulation intensity eliciting maximal quadriceps twitch and M_{MAX} with increasing stimulus intensities. No electrical activity of the biceps femoris (BF) was observed during stimulation. For the evaluation of the quadriceps function, potentiated twitches force were measured after each 3 s maximal isometric voluntary contraction (MVC). Six MVCs, separated by 1 min, were performed prior to the time-trials and the average of the best three MVCs was used as pre-exercise baseline. One MVC was performed at 10 s, 1 min, 2 min, 4 min, 6 min and 15 min after exercise termination.

Potentiated twitch force evoked by single electrical stimulation of the femoral nerve (QT_{Single}), and paired electrical stimulations at a frequency of 10 Hz (QT_{10}) and 100 Hz (QT_{100}) were elicited 3 s, 6 s, and 9 s after each MVC, respectively. For all QT_{10} , QT_{100} , and MVCs, we determined peak force. For all QT_{Single} , peak force, contraction time to peak force (CT), maximal rate of force development (MRFD, maximal value of the first derivative of the force signal) and half relaxation time (HRT, time to obtain half of the decline in maximal force) were assessed. Interpolated 100Hz-paired stimuli were delivered during the peak force of each MVC to determine VA of the quadriceps (23). Voluntary activation was calculated according to the following formula: $VA (\%) = (1 - QT_{100,interpolated} / QT_{100}) \times 100$, where $QT_{100,interpolated}$ is the size of the interpolated twitch force.

Quadriceps (peripheral) fatigue was calculated as the percent difference in evoked force from pre- to post-exercise (ΔQT_{Single} , ΔQT_{10} , and ΔQT_{100}) and expressed as a percent change from pre-exercise. The ratio QT_{10} / QT_{100} ($QT_{10/100}$) was calculated as a decrease in this ratio is commonly interpreted as an index of low-frequency fatigue (22).

Surface electromyography

Electrical activity of the VL, the VM and the BF of the right leg was recorded by three pairs of Ag/AgCl surface electrodes (diameter = 10 mm; inter-electrode distance = 20 mm; Mini-KR, Contrôle-Graphique, Brie-Comte-Robert, France) placed on the muscle belly connected to an EMG system (Octal Bio-Amp, ML138, ADInstrument, Bella-Vista, Australia). A reference electrode was placed on the right lateral tibial condyle. The skin was shaved, abraded with emery paper and cleaned with alcohol to reduce skin impedance below 3 k Ω (i.e. 2.1 ± 0.3 k Ω). The position of the electrodes optimizing M_{MAX} was marked with indelible ink to ensure identical placement at subsequent visits. EMG signals were amplified, filtered (bandwidth frequency, 0.03 Hz – 1 kHz), and recorded (sampling frequency, 4 kHz) using commercially available software (Labchart 7, ADInstruments, Bella-Vista, Australia). Using a custom-made Matlab (Matlab 7.12, MathWorks, Natick, MA, USA) algorithm, each burst onset and offset of the rectified EMG signal, recorded during the time-trials,

was determined. During exercise, crank angle was monitored continuously and the root mean square (RMS) of the EMG signal recorded during each time-trial was calculated over 50ms time windows surrounding a crank angle of 45° clockwise, during the peak cycling EMG burst of the VL and VM (32). The RMS recorded during exercise was then normalized to the RMS recorded during pre-exercise MVC ($\text{RMS}_{\%MVC}$), and averaged every 500m. RMS during each MVC was calculated as the average value over a 0.5-s interval during the plateau phase of the MVC. Many factors can influence the bipolar EMG signal, which warrants caution when inferring motoneuronal activity from surface EMG (19). However, the concomitant augmentation of the EMG signal and power output during the initial phase of exercise in our different conditions, a time period that is characterized by little peripheral fatigue, supports the validity of $\text{RMS}_{\%MVC}$ as an estimate of muscle activation in the current study.

Metabolic and cardio-ventilatory indices.

Pulmonary ventilation (minute ventilation, \dot{V}_E ; breathing frequency, f_B ; tidal volume, V_T) and gas exchange (oxygen uptake, $\dot{V}O_2$; CO_2 production, $\dot{V}CO_2$; respiratory exchange ratio, RER) indices were measured breath-by-breath at rest and throughout the time-trials using a stationary automatic ergospirometer (MS-CPX, Viasys, San Diego, California, USA). Before each test, gas analysers were calibrated using a certified gas preparation (O_2 : 16% - CO_2 : 5%) and an accurate volume of ambient air (2L) was used to adjust the pneumotachograph. Heart rate was calculated from R-R intervals recorded at 1kHz by a heart rate monitor (RS800CX, Polar Electro, Kempele, Finland). Capillary blood lactate samples (5 μ l) were collected from a fingertip at rest and 3 min post-exercise. Blood samples were immediately analysed with a test strip by an electrochemical method (LactatePro, Arkray, Kyoto, Japan) for determination of capillary blood lactate concentration ($[La]_b$).

Psychological indices

Rating of perceived exertion (RPE), internal attentional focus and self-efficacy were recorded during each session at every kilometer. To evaluate RPE, subjects were asked to rate on the centiMax scale (CR100, 7) how hard, heavy and strenuous was the exercise during the preceding kilometer. This scale ranged from “0 - Nothing at all” - to “100 - Maximal”. The anchoring for “100 - maximal” was the maximal effort experienced during the fastest 5km time-trial during the practice sessions.

To evaluate internal attentional focus, participants were asked to indicate on a 20 cm scale for how long their attention was focused towards internal inputs such as leg fatigue, pain, heart beating or breathing frequency and depth. This scale was adapted from the 4th item of the body vigilance scale (31) and ranged from 0 – “Never” to 100 – “all the time”. To evaluate self-efficacy,

participants were asked to rate on a 20 cm scale their ability to continue at the current pace for the remaining distance of each 5 km cycling time-trial (36, 37).

Statistical analysis

Data presented in the results section are expressed as mean \pm SD. Data presented in the figures are expressed as mean \pm SEM. Normality of every dependent variable and homogeneity of the variance of the distributions (equal variance) were confirmed using the Kolmogorov-Smirnov test and the Levene test, respectively. To protect against the risk of type I error arising from multiple comparisons (35), a multivariate analysis (MANOVA) was conducted on our dependent variables recorded during exercise (i.e. power output, $\text{RMS}_{\%MVC}$, psychological variables) or during post-exercise recovery (i.e. neuromuscular fatigue indices). A significant ($p < 0.001$) condition \times time effect was found for both the exercise and post-exercise recovery dataset. Then, two-way ANOVAs with repeated measures (condition \times time) were used to test for condition effect across time on power output, $\text{RMS}_{\%MVC}$, psychological variables during exercise, and fatigue indices during post-exercise recovery. Bonferroni correction was used to eliminate false positives derived from multiple comparisons. When a significant difference was found with the two-way ANOVA, multiple comparisons analysis was performed using the Tukey’s HSD test. In addition, a one-way ANOVA with repeated measures were used to determine differences across conditions on absolute values of time-to-completion, mean power output, $\dot{V}O_2$, $\dot{V}CO_2$, \dot{V}_E , $\dot{V}_E \cdot \dot{V}CO_2$, f_B , V_T , RER and $[La]_b$ averaged over the entire cycling time-trial. Effect size was assessed using partial eta-squared (η^2). A η^2 index for effect size was considered as small when η^2 was close to 0.02, as medium when η^2 was close to 0.13 and as large when η^2 was close to 0.26 (10). Student’s paired t test was used to determine differences in performance and neuromuscular variables between the best time familiarization session and 5K_{CTRL}, and on performance indices and $\text{RMS}_{\%MVC}$ between 5K_{100%} and 5K_{105%} first and second half. Effect size was then assessed using the Cohen’s d index. A Cohen’s d index for effect size was considered as small when d was close to 0.2, as medium when d was close to 0.5 and as large when d was close to 0.8 (10). For assessment of the within-session and/or between-sessions reliability of our data recorded during exercise and during neuromuscular testing, we tested for differences between the best practice time-trial and 5K_{CTRL} using Student’s paired t test and calculated standard error as well as intraclass correlation coefficients (ICC) as previously recommended (16). Reliability was considered excellent when ICC was > 0.75 , good when ICC was < 0.75 and > 0.60 , fair when ICC was < 0.60 and > 0.40 and poor when ICC was < 0.40 (9). For qualitative analysis purpose, the method of Hopkins (15) was used to determine for males and females the estimated worthwhile meaningful improvement in time to

complete a cycling time-trial. We considered that a subject improved his/her performance when completion time was shortened beyond the estimated worthwhile meaningful improvement. For a given variable, percent change from one condition to another was calculated as follow: % change (%) = $(\text{value}_{\#1} - \text{value}_{\#2}) / \text{value}_{\#2} \times 100$, where $\text{value}_{\#1}$ and $\text{value}_{\#2}$ represent values from two different experimental conditions or from two different time within a given experimental condition (e.g. $\text{value}_{\#1}$: mean power output in 5K_{102%}; $\text{value}_{\#2}$: mean power output in 5K_{100%}). Statistical analyses were conducted using Statistica 8.0 (StatSoft, Inc., Tulsa, OK, US). Statistical significance was set at $p < 0.05$.

RESULTS

Performance and neuromuscular variables reproducibility

Completion time (9.17 ± 1.22 min vs. 9.16 ± 1.20 min, $p = 0.94$, $d = 0.03$) and mean power output (217 ± 59 W vs. 218 ± 62 W, $p = 0.80$, $d = 0.06$) were not significantly different between the best practice time-trial and 5K_{CTRL}. “Excellent” reliability for mean power output (CV = $1.7 \pm 1.8\%$, ICC = 0.990) and completion time (CV = $0.9 \pm 0.8\%$, ICC = 0.990) were also found. From these baseline data, the estimated worthwhile meaningful improvement in time to complete a cycling time-trial was 0.79 % for males (i.e. representative of 4.4 s) and 0.62 % for females (i.e. representative of 3.4 s).

For the neuromuscular variables, no significant within-session or between-session difference was found and “excellent” reliability was shown for baseline MVC force ($p > 0.41$, CV < 4.7 %, ICC > 0.958), VA ($p > 0.51$, CV < 2.4 %, ICC > 0.763), QT_{single} ($p > 0.45$, CV < 3.8 %, ICC > 0.957), QT₁₀ ($p > 0.35$, CV < 4.4 %, ICC > 0.959) and QT₁₀₀ ($p > 0.62$, CV < 3.4 %, ICC > 0.958).

Effects of deceptive time-trials on exercise performance and quadriceps neuromuscular function.

Exercise performance and quadriceps EMG. The changes of mean power output as well as EMG during the 5 km cycling time-trials performed with augmented deceptive feedback are shown in Figure 1. Mean group and individual performance indices are shown in Figure 2. For all trials, no significant exercise-induced alteration in M_{MAX} was found for both VL and VM, demonstrating no alteration in membrane excitability during time-trials (Table 1).

When the exercise data from every experimental visits were pooled together, a significant condition \times exercise duration effect was found for power output ($p < 0.001$, $\eta^2 = 0.40$) and RMS_{%MVC} ($p < 0.001$, $\eta^2 = 0.23$). Post-hoc analysis revealed that no significant difference in RMS_{%MVC} (VL: $57 \pm 18\%$ vs. $61 \pm 24\%$; $p = 0.37$; VM: $56 \pm 25\%$ vs. $60 \pm 24\%$; $p = 0.53$), mean power output (218 ± 60 W vs. 219 ± 61 W; $p = 0.95$) and completion time (9.16 ± 1.14 min vs. 9.16 ± 1.20 min; $p = 0.98$) was observed between 5K_{100%} and 5K_{CTRL}. No significant

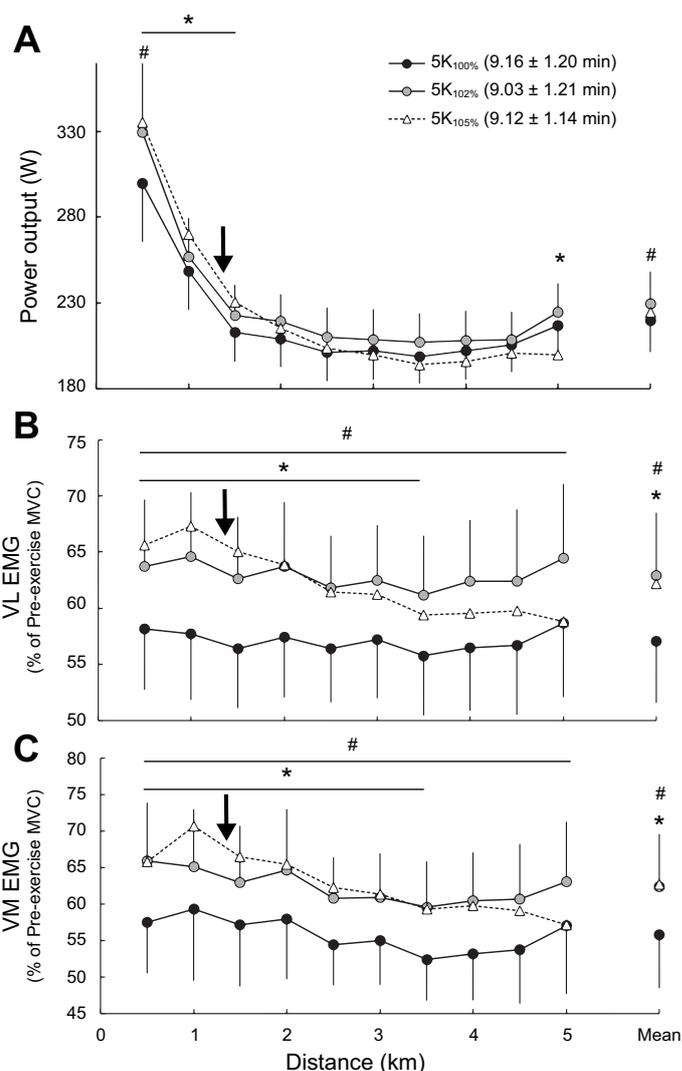


Figure 1. Power output (A), vastus lateralis (VL, B) and vastus medialis (VM, C) EMG activity responses to different magnitudes of deception during a 5km cycling time-trial.

5K_{100%}, 5K_{102%} and 5K_{105%} represent the 5 km cycling time-trials during which subjects followed a simulated dynamic avatar reproducing 100%, 102% and 105% of their previous fastest 5 km time-trial without a virtual pacemaker (i.e. 5K_{CTRL}), respectively. The root mean square (RMS) of the EMG signal recorded during each time-trial was normalized to the RMS recorded during pre-exercise maximal voluntary isometric contraction (RMS_{%MVC}). The black arrow represents the mean distance at which participants were not able to follow the virtual pacemaker anymore during 5K_{105%}. # significant difference between 5K_{100%} and 5K_{102%} ($p < 0.05$); * significant difference between 5K_{100%} and 5K_{105%} ($p < 0.05$). Results are presented as mean \pm SEM.

difference in RMS_{%MVC} ($p = 0.87$) and mean power output ($p = 0.98$) was also found at a given kilometer between 4K_{100%} and 5K_{100%}. During 5K_{102%}, all subjects improved performance time and power output. Group mean VL and VM RMS_{%MVC}, power output and performance time were improved compared to 5K_{100%} by

INCREASED FATIGUE AFTER DECEPTIVE TIME-TRIAL

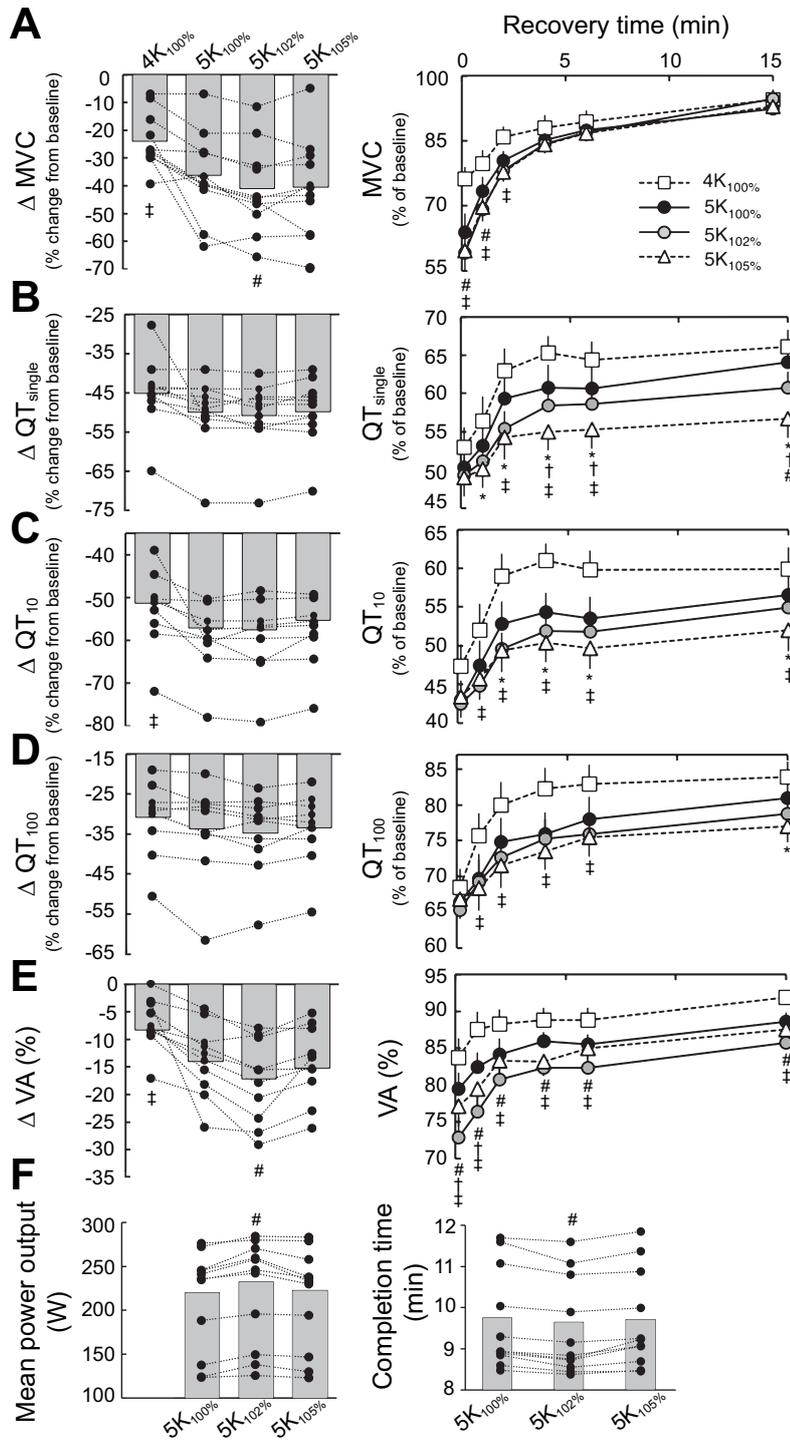


Figure 2. Performance and fatigue responses to different magnitudes of deception during a 5km cycling time-trial. 5K_{100%}, 5K_{102%} and 5K_{105%} represent the 5 km cycling time-trials during which subjects followed a simulated dynamic avatar reproducing 100%, 102% and 105% of their previous fastest 5 km time-trial without a virtual pacemaker (i.e. 5K_{CTRL}), respectively. 4K_{100%} represents the cycling time-trial during which subjects followed a simulated dynamic avatar reproducing 100% of their previous fastest 5km time-trial without a virtual pacemaker (i.e. 5K_{CTRL}) but stopped exercise at 4 km. Group mean (grey bars) and individual (black round & dotted line) data for fatigue indices (left panels) are presented as pre- to immediate post-exercise (i.e. 10 s) reduction in maximal voluntary isometric contraction of quadriceps force (MVC, A), potentiated twitch force evoked by single (QT_{single}, B), 10 Hz-paired (QT₁₀, C), and 100 Hz-paired (QT₁₀₀, D) electrical stimulation of the femoral nerve, and voluntary activation of the quadriceps (VA, E). Group mean and individual data are also presented for mean power output (F, left panel) and time-to-completion (F, right panel). MVC, QT_{single}, QT₁₀ and QT₁₀₀ during recovery (from 10s through 15min post-exercise, right panels) are presented as percent of baseline whereas VA is presented as %. # significant difference between 5K_{100%} and 5K_{102%} ($p < 0.05$); * significant difference between 5K_{100%} and 5K_{105%} ($p < 0.05$); † significant difference between 5K_{102%} and 5K_{105%} ($p < 0.05$); ‡ significant difference between 4K_{100%} and 5K_{100%} ($p < 0.05$). Data are presented as mean \pm SEM.

INCREASED FATIGUE AFTER DECEPTIVE TIME-TRIAL

Table 1. Effect of different magnitudes of augmented deceptive feedback during cycling time-trials on the neuromuscular function

	5K _{CTRL}			5K _{100%}			5K _{102%}			5K _{105%}			4K _{100%}		
	Pre	10s		Pre	10s	15min	Pre	10s	15min	Pre	10s	15min	Pre	10s	15min
MVC (N)	563 ± 124	350 ± 98 \$		593 ± 135	379 ± 119 \$	568 ± 136	585 ± 124	338 ± 106 \$,*	575 ± 140	580 ± 135	348 ± 128 \$	562 ± 143	581 ± 134	444 ± 120 \$,*	553 ± 147
VA (%)	92 ± 3	79 ± 9 \$		92 ± 1	79 ± 7 \$	89 ± 4	90 ± 3	73 ± 9 \$ *	86 ± 5\$	92 ± 2	77 ± 9 \$,†	88 ± 6	92 ± 2	84 ± 9 \$,*	92 ± 3 *
QT _{sgl} (N)	171 ± 27	85 ± 24 \$		173 ± 28	87 ± 20 \$	107 ± 7 \$	167 ± 28	81 ± 18 \$	105 ± 7 \$,*	171 ± 29	84 ± 21 \$	101 ± 9 \$,* ,†	168 ± 30	88 ± 20 \$	108 ± 24 \$
QT ₁₀ (N)	268 ± 51	115 ± 34 \$		276 ± 61	117 ± 27 \$	154 ± 12 \$	264 ± 60	110 ± 23 \$	144 ± 12 \$	272 ± 57	116 ± 30 \$	142 ± 13 \$,*	269 ± 56	125 ± 29 \$	160 ± 38 *
QT ₁₀₀ (N)	250 ± 39	174 ± 35 \$		256 ± 44	168 ± 29 \$	196 ± 12 \$	254 ± 43	164 ± 26 \$	198 ± 9 \$	256 ± 44	169 ± 29 \$	195 ± 11 \$,*	251 ± 43	170 ± 25 \$	209 ± 29
QT _{10/100} Ø	1.07 ± 0.04	0.65 ± 0.07 \$		1.07 ± 0.06	0.69 ± 0.09 \$	0.75 ± 0.11 \$	1.03 ± 0.08	0.67 ± 0.12 \$	0.72 ± 0.11 \$	1.06 ± 0.06	0.68 ± 0.11 \$	0.71 ± 0.10 \$	1.06 ± 0.06	0.73 ± 0.09 \$	0.76 ± 0.11 \$
MRFD (N.ms ⁻¹)	5.7 ± 1.2	2.9 ± 0.8 \$		5.7 ± 1.0	2.9 ± 0.7 \$	3.4 ± 0.3 \$	5.1 ± 0.8	2.5 ± 0.4 \$	3.4 ± 0.2 \$	5.4 ± 1.1	2.7 ± 0.7 \$	3.3 ± 0.3 \$	5.3 ± 0.9	2.9 ± 0.7 \$	3.6 ± 0.8 \$
CT (ms)	61 ± 13	45 ± 9 \$		66 ± 15	45 ± 7 \$	50 ± 4 \$	67 ± 12	48 ± 7 \$	46 ± 4 \$	64 ± 14	46 ± 8 \$	46 ± 4 \$	64 ± 14	47 ± 10 \$	50 ± 12 \$
HRT (ms)	76 ± 8	82 ± 16 \$		72 ± 6	90 ± 15 \$	70 ± 4	70 ± 6	85 ± 11 \$	69 ± 4	71 ± 7	84 ± 9 \$	67 ± 4 \$	70 ± 6	87 ± 12 \$	61 ± 8 \$
VL M _{MAX} (mV)	14 ± 5	14 ± 5		13 ± 5	14 ± 5	13 ± 5	14 ± 5	15 ± 5	14 ± 5	14 ± 5	15 ± 5	14 ± 5	14 ± 5	15 ± 5	14 ± 5
VM M _{MAX} (mV)	20 ± 4	20 ± 4		21 ± 3	21 ± 4	21 ± 5	20 ± 4	20 ± 4	20 ± 4	20 ± 6	21 ± 6	20 ± 6	21 ± 4	21 ± 4	20 ± 4

MVC, maximal voluntary isometric contraction; VA, voluntary activation of the quadriceps; QT_{single}, potentiated quadriceps twitch evoked by single electrical stimulation of the femoral nerve, QT₁₀, potentiated quadriceps twitch evoked by 10 Hz-paired electrical stimulation of the femoral nerve, QT₁₀₀, potentiated quadriceps twitch evoked by 100 Hz-paired electrical stimulation of the femoral nerve; QT_{10:100}, low-frequency fatigue ratio; MRFD, maximal rate of force development; CT, contraction time; HRT, half relaxation time; M_{MAX}, maximal amplitude of the compound muscle action potential; VL, *vastus lateralis*; VM, *vastus medialis*; 5K_{100%}, 5K_{102%} and 5K_{105%} represent the 5 km cycling time trials during which subjects followed a simulated dynamic avatar reproducing 100%, 102% and 105% of their previous fastest 5 km time trial without a virtual pacemaker (i.e. 5K_{CTRL}), respectively. 4K_{100%} represents the cycling time trial during which subjects followed a simulated dynamic avatar reproducing 100% of their previous fastest 5km time trial without a virtual pacemaker (i.e. 5K_{CTRL}) but stopped exercise at 4 km. Neuromuscular assessment was interrupted 6 min after the end of the time trial in 5K_{CTRL} (vs. 15 min in the other experimental conditions) because subject had to perform another test (data not shown). No significant difference was found for baseline measurement between conditions. \$ significant difference compared to pre-exercise value ($p < 0.05$); * significant difference compared to 5K_{100%} ($p < 0.05$); † significant difference between 5K_{102%} and 5K_{105%} ($p < 0.05$).

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12 ± 18 % ($p < 0.001$) and 15 ± 26 % ($p < 0.05$), 5 ± 2 % ($p < 0.01$) and 2 ± 1 % ($p < 0.001$), respectively.

During 5K_{105%}, subjects were able to follow the virtual pacemaker up to an average of 1530 ± 1387 m (range: 450 – 2500). During the first half of the time-trial, VL and VM RMS_{%MVC} were 14 ± 17% ($p < 0.05$, $d = 0.68$) and 18 ± 16 % ($p < 0.001$, $d = 0.93$) greater in 5K_{105%} compared to 5K_{100%} (Fig. 1). This higher surface EMG amplitude during the first half of the 5K_{105%} time-trial occurred together with a 7 ± 5 % higher power output ($p < 0.01$, $d = 1.00$) and a 2 ± 2 % faster completion time ($p < 0.01$, $d = 1.01$) compared to 5K_{100%} (Fig. 1). During the second half of the time-trial, mean power output (18 ± 12%, $p < 0.001$, $d = 1.23$) and completion time 4 ± 6% ($p < 0.05$, $d = 0.69$) were significantly reduced and increased in 5K_{105%}, respectively. Over the entire time-trial, group mean average VL and VM RMS_{%MVC} was 10 ± 16 % and 15 ± 17 % greater in 5K_{105%} compared to 5K_{100%} ($p < 0.05$), whereas mean power output (223 ± 60 vs. 219 ± 61 W; $p = 0.25$) and completion time (9.12 ± 1.2 min and 9.16 ± 1.14 min; $p = 0.59$) were similar between conditions.

Neuromuscular fatigue indices. Group mean and individual exercise-induced changes in peripheral and central fatigue variables are shown in Figure 2. Group mean raw values for the neuromuscular indices are presented in Table 1. In all subjects, potentiated twitch

force was significantly reduced following every time-trial. There was evidence of substantial peripheral fatigue as documented by exercise-induced reductions in QT_{Single}, QT₁₀, QT₁₀₀ and QT_{10/100} ($p < 0.001$, $\eta^2 > 0.73$), all of which persisted for at least 15 minutes upon completion of every time-trials. Similarly, VA, MVC force, MRFD, and HRT were also significantly attenuated after every time-trial ($p < 0.01$).

When the pre- to post-exercise neuromuscular data from every experimental visit were pooled together, a significant condition × recovery duration effect was found ($p < 0.001$, $\eta^2 > 0.62$). Specifically, 10 s after the completion of the time-trial, the exercise-induced alterations in MVC force and VA were 14 ± 19 % (-41 ± 15 % vs. -36 ± 15 %; $p < 0.05$) and 28 ± 31 % greater (-18 ± 7 % vs. -14 ± 6 %; $p < 0.05$) in 5K_{102%} compared to 5K_{100%} (Fig. 2). However, no significant difference in Δ QT_{Single} ($p = 0.99$), Δ QT₁₀ ($p = 0.99$) and Δ QT₁₀₀ ($p = 0.99$) were found between conditions. No significant difference in MVC force ($p = 0.13$), Δ VA ($p = 0.94$), Δ QT_{Single} ($p = 0.99$), Δ QT₁₀ ($p = 0.99$) and Δ QT₁₀₀ ($p = 0.99$) were found between 5K_{105%} and 5K_{100%}. In 4K_{100%}, exercise induced reduction in MVC force, VA and Δ QT₁₀ were 34 ± 23 % ($p < 0.01$), 39 ± 40 % ($p < 0.01$) and 8 ± 13 % ($p < 0.01$) less compared to 5K_{100%}, whereas Δ QT_{Single} ($p = 0.35$) and Δ QT₁₀₀ ($p = 0.92$) and Δ QT_{10/100} ($p = 0.14$) were not significantly different.

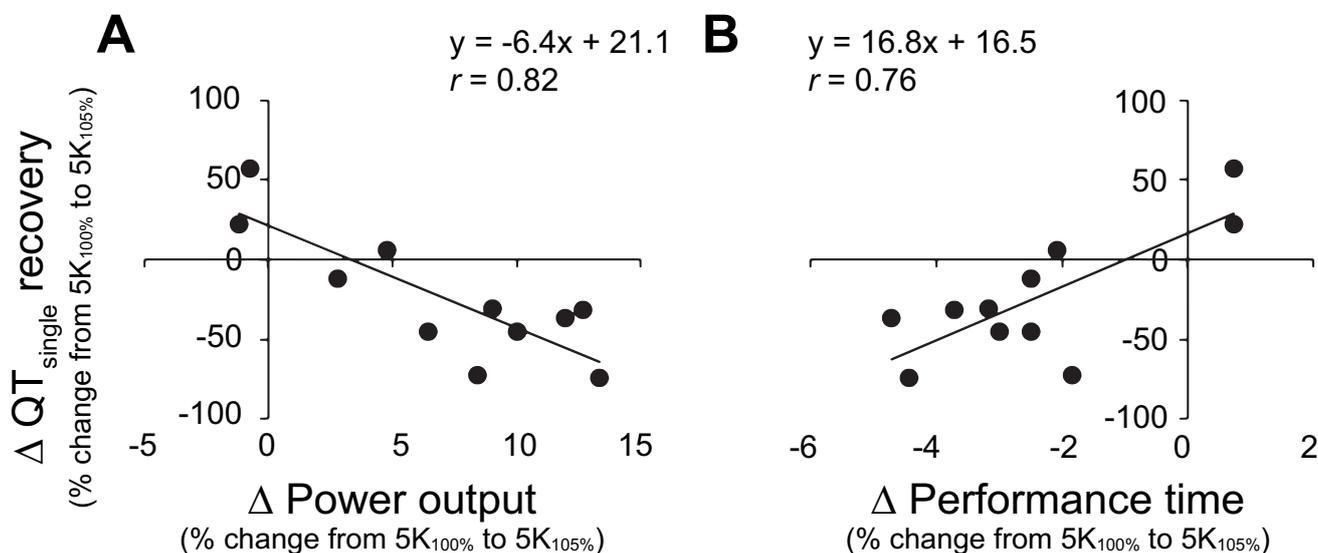


Figure 3. Relationship between potentiated twitch force recovery and performance indices.

5K_{100%} and 5K_{105%} represent the 5 km cycling time-trials during which the subject followed a simulated dynamic avatar reproducing 100% and 105% (5K_{105%}) of his previous fastest 5 km cycling time-trial without a virtual pacemaker (i.e. 5K_{CTRL}), respectively. Δ QT_{single} recovery represents the % change in QT_{single} from 10 s to 15 min following exercise termination, from 5K_{100%} to 5K_{105%}. Δ Power output and Δ Performance time represent the % change during the first half of the time-trials in power output and completion time, from 5K_{100%} to 5K_{105%}, respectively

Fifteen minutes after the completion of the time-trial, partial recovery was observed in all fatigue indices (Fig. 2). MVC force and VA were not significantly different from baseline values in all conditions ($p > 0.49$),

although VA remained slightly reduced in 5K_{102%} compared to 5K_{100%} ($p < 0.05$). In contrast, evoked potentiated twitches force remained significantly depressed ($p < 0.05$) and this impairment in muscle force

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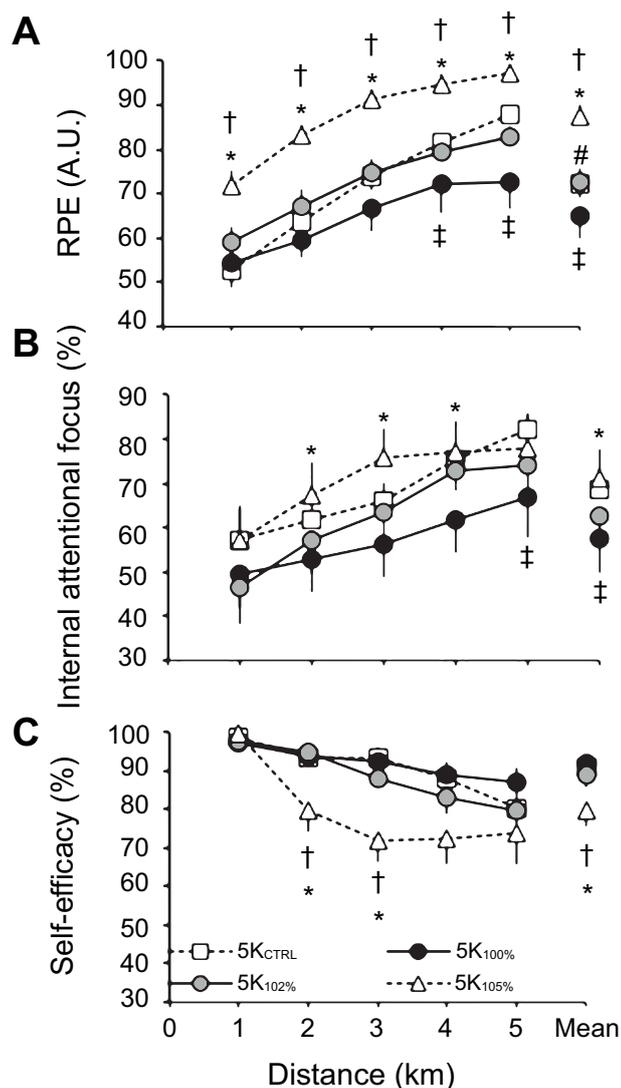


Figure 4. Influence of different magnitudes of deception during cycling time-trials on psychological indices during 5km cycling time-trial.

5K_{100%}, 5K_{102%} and 5K_{105%} represent the 5 km cycling time-trials during which subjects followed a simulated dynamic avatar reproducing 100%, 102% and 105% of their previous fastest 5 km time-trial without a virtual pacemaker (i.e. 5K_{CTRL}), respectively. Rate of perceived exertion (RPE, A), internal attentional focus (B) and self-efficacy (C) were assessed every kilometer using analogic scales. ‡ significant difference between 5K_{100%} and 5K_{CTRL} ($p < 0.05$); † significant difference between 5K_{105%} and 5K_{CTRL}; # significant difference between 5K_{100%} and 5K_{102%} ($p < 0.05$); * significant difference between 5K_{100%} and 5K_{105%} ($p < 0.05$).

recovery was more pronounced in 5K_{105%}. Specifically, after 5K_{105%}, QT_{Single}, QT₁₀ and QT₁₀₀ recovery was 11 ± 10 %, 8 ± 11 % and 5 ± 7 % less than 5K_{100%}, respectively ($p < 0.01$), whereas QT_{Single} recovery was also 7 ± 8 % less than 5K_{102%}. In addition, QT_{Single} recovery was 5 ± 5 % less in 5K_{102%} than in 5K_{100%}. When the potentiated twitch force data from 10 s to fifteen minutes post-exercise were pooled together, reduction in Δ QT_{Single}, Δ QT₁₀ and Δ QT₁₀₀ were 6 ± 9 %, 4 ± 9 % and 6 ± 8 % ($p < 0.05$) more pronounced after

5K_{102%} compared to 5K_{100%}, and 11 ± 9 %, 6 ± 6 % and 8 ± 21 % ($p < 0.05$) after 5K_{105%} compared to 5K_{100%}, respectively. Finally, as illustrated in Fig. 3, the percent change in QT_{Single} recovery (i.e. from 10 s to 15 min post-exercise) from 5K_{100%} to 5K_{105%} was correlated with the % change in power output and completion time during the first half of the time-trial from 5K_{100%} to 5K_{105%}.

Perceived exertion and internal attentional focus.

When the psychological variables measured during exercise from every experimental visits were pooled together, a significant condition \times exercise duration effect was found ($p < 0.001$, $\eta^2 > 0.14$). In all subjects, RPE and internal attentional focus gradually increased whereas self-efficacy gradually decreased over the course of the time-trial in every condition ($p < 0.001$, Fig. 4). In 5K_{100%}, mean RPE and mean internal attentional focus were reduced by 10 ± 22 % and 17 ± 32 % compared to 5K_{CTRL}, respectively ($p < 0.001$). In 5K_{102%}, mean RPE was 21 ± 47 % greater ($p < 0.05$) compared to 5K_{CTRL} ($p > 0.87$). No difference was found in self-efficacy between 5K_{CTRL}, 5K_{100%} and 5K_{102%} ($p > 0.93$). In 5K_{105%}, mean RPE increased by 23 ± 13 %, 54 ± 87 % and 21 ± 19 % compared to 5K_{CTRL}, 5K_{100%} and 5K_{102%}, respectively ($p < 0.01$). Moreover, mean internal attentional focus was increased by 23 ± 19 % compared to 5K_{100%} whereas self-efficacy was reduced by 12 ± 14 % and 14 ± 14 % compared to 5K_{CTRL} and 5K_{100%}, respectively ($p < 0.05$).

Metabolic and cardio-ventilatory indices.

Group mean metabolic and cardio-ventilatory data collected before, during and following the cycling time-trials are summarized in Table 2. A significant condition effect was found for $\dot{V}O_2$ ($p < 0.001$, $\eta^2 = 0.48$), $\dot{V}CO_2$ ($p < 0.001$, $\eta^2 = 0.59$), $\dot{V}E$ ($p < 0.001$, $\eta^2 = 0.48$), f_B ($p < 0.001$, $\eta^2 = 0.52$) and HR ($p < 0.05$, $\eta^2 = 0.29$). In contrast, no significant condition effect was found for V_T ($p = 0.67$, $\eta^2 = 0.06$), $\dot{V}CO_2 \cdot \dot{V}O_2^{-1}$ ($p = 0.65$, $\eta^2 = 0.02$), $\dot{V}E \cdot \dot{V}CO_2^{-1}$ ($p = 0.36$, $\eta^2 = 0.10$) and $[La]_b$ ($p = 0.08$, $\eta^2 = 0.20$). Specifically, $\dot{V}O_2$ ($p = 0.70$), $\dot{V}CO_2$ ($p = 0.35$), $\dot{V}E$ ($p = 0.99$), f_B ($p = 0.93$) and HR ($p = 0.40$) were similar in 5K_{100%} compared to 5K_{CTRL}. In 5K_{102%}, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, f_B and HR were 2 ± 2 % ($p < 0.01$), 6 ± 4 % ($p < 0.001$), 6 ± 4 % ($p < 0.001$), 8 ± 6 % ($p < 0.001$) and 2 ± 2 % ($p < 0.05$) higher compared to 5K_{100%}, respectively. In 5K_{105%}, $\dot{V}CO_2$, $\dot{V}E$, and BF were 4 ± 4 % ($p < 0.001$), 4 ± 5 % ($p < 0.05$) and 5 ± 4 % ($p < 0.05$) higher compared to 5K_{100%}, respectively. In 4K_{100%}, $[La]_b$ (10.8 ± 2 mmol.L⁻¹) was 11 ± 17 % lower than 5K_{100%} ($p < 0.05$).

Table 2. Average metabolic and cardio-ventilatory responses to deceptive cycling time trial.

Variable	Baseline	5K _{CTRL}	5K _{100%}	5K _{102%}	5K _{105%}
$\dot{V}O_2$ (ml.min ⁻¹ .kg ⁻¹)	7 ± 2	42 ± 8	42 ± 8	43 ± 8 *	42 ± 8
$\dot{V}CO_2$ (ml.min ⁻¹ .kg ⁻¹)	6 ± 2	42 ± 7	41 ± 8	43 ± 8 *	43 ± 8 *
$\dot{V}CO_2 \cdot \dot{V}O_2^{-1}$ (\emptyset)	0.9 ± 0.1	1.00 ± 0.03	0.99 ± 0.04	1.01 ± 0.05	1.01 ± 0.03
\dot{V}_E (L.min ⁻¹)	14 ± 4	94 ± 21	94 ± 21	99 ± 23 *	98 ± 23 *
$\dot{V}_E \cdot \dot{V}CO_2^{-1}$ (\emptyset)	2.2 ± 0.3	2.2 ± 0.2	2.3 ± 0.2	2.3 ± 0.3	2.3 ± 0.3
f_B (breaths.min ⁻¹)	14 ± 4	42 ± 3	42 ± 4	46 ± 5 *	44 ± 5 *
V_T (L)	1.1 ± 0.3	2.3 ± 0.6	2.3 ± 0.6	2.2 ± 0.5	2.3 ± 0.6
HR (beats.min ⁻¹)	76 ± 9	172 ± 5	170 ± 4	174 ± 6 *	170 ± 4
[La] _b (mmol.L ⁻¹)	1.5 ± 0.3	12.8 ± 3	11.9 ± 3	12.7 ± 3	12.4 ± 3

$\dot{V}O_2$, oxygen uptake; $\dot{V}CO_2$, CO₂ production; \dot{V}_E , minute ventilation; f_B , breathing frequency; V_T , tidal volume; HR, heart rate, [La]_b, capillary blood lactate. 5K_{100%}, 5K_{102%} and 5K_{105%} represent the 5 km cycling time trials during which subjects followed a simulated dynamic avatar reproducing 100%, 102% and 105% of their previous fastest 5 km time trial without a virtual pacemaker (i.e. 5K_{CTRL}), respectively. Baseline data are expressed as the mean value of pre-exercise data from all four experimental visits. Exercise data are expressed as the mean values over the entire 5 km cycling time trial. No significant difference was found between 5K_{CTRL} and 5K_{100%}. * significant difference from 5K_{100%} ($p < 0.05$).

DISCUSSION

This study assessed the neuromuscular consequences and underpinning performance improvement during deceptive cycling time-trials. Improved muscle activation, power output, metabolic and cardio-ventilatory responses, and completion time during deceptive time-trial was achieved only when the magnitude of deception was moderate (i.e. 2% greater speed, corresponding to 5% greater power output) and was associated with a significant reduction in participants' ability to produce force. Specifically, a further impairment in maximal voluntary contraction force, voluntary activation and potentiated twitch force recovery were found with deception compared to control. Our data are the first to demonstrate that performance improvement with augmented deceptive feedback results in exacerbated central and peripheral fatigue following cycling time-trial.

Why did a moderate magnitude of deception improve time-trial performance and what were the consequences on the neuromuscular function?

Compared to a self-paced time-trial, a significant increase in muscle activation was found during deceptive time-trial performed with moderate magnitude of deception (i.e. 2% greater speed). This

greater muscle activation with deception, which is thought to reflect additional recruitment of motor units and/or increased firing frequency (26), likely enabled subjects to cycle at a higher power output (+ 5%) and to improve completion time (- 2%). This increase in power output occurred together with a small but significant increase in the metabolic and cardio-ventilatory responses to exercise in 5K_{102%}, indicative of a greater metabolic work. Performance improvements with augmented deceptive feedback led to a greater exercise-induced reduction in participants' ability to produce force compared to self-paced time-trial, as evidenced by the ~14 % greater reduction in MVC force in 5K_{102%} compared to 5K_{CTRL}. Moreover, a greater alteration in potentiated twitch force recovery as well as a larger reduction in voluntary activation were found post-exercise in 5K_{102%} compared to 5K_{CTRL}. This further increase in exercise-induced fatigue in 5K_{102%} was therefore accounted for by mechanisms from both peripheral and central origins.

The increases in power output and EMG amplitude, indicative of higher muscle activation, in 5K_{102%} compared to 5K_{100%} were associated with a impairment of quadriceps force recovery (i.e. from 10 s to 15 min post-exercise; see below). However, the absence of a further reduction in the potentiated twitch force at exercise termination (i.e. 10 s post-exercise) despite a higher power output and metabolic demand in 5K_{102%} compared to 5K_{100%} seems to contradict the understanding that peripheral fatigue is closely related to intramuscular metabolite concentration (1, 6). Specifically, recent findings from Blain et al. (6) showed a linear relationship between the increased concentrations of fatigue-related metabolites (e.g. inorganic phosphate, H⁺) and the increased peripheral fatigue following a 5 km cycling time-trial performed with attenuated group III-IV muscle afferents (using intrathecal fentanyl) vs. with intact muscle afferent feedback. Although potentiated twitch force evoked with electrical stimulation at different frequencies has been shown to be effective in detecting small degree of exercise-induced muscle fatigue (20), this variable might not have been sensitive enough to reveal the effect of a five percent improvement in power output on peripheral fatigue in 5K_{102%} compared to 5K_{100%}. Alternatively, while intramuscular metabolic perturbation was substantially increased with fentanyl compared to the control condition in Blain's study (6), the small increase in metabolic demand (and associated muscle metabolites) in 5K_{102%} compared to 5K_{100%} may not have been sufficient to cause further significant depressive effects on the quadriceps contractile function right at exercise termination. The higher power output and metabolic work may have rather altered processes involved in quadriceps force recovery (see below). This hypothesis is consistent with our results comparing potentiated twitch force in 4K_{100%} vs. 5K_{100%}. Our findings indeed showed that the 8% increase in power output and the 11% increase [La]_b (indicative of greater

metabolic disturbance) during the last kilometer of 5K_{100%} had little effect on exercise-induced peripheral fatigue measured at exercise termination but significantly impaired potentiated twitch force recovery (Fig. 2). Our hypothesis is also consistent with isolated muscle fibers data showing that the inhibitory effect of additional metabolites during a state of high concentration of metabolites affects muscle force to a small magnitude (25, 29).

Why didn't time-trial performance improve with large magnitude of deception?

In contrast to deception of 2 % greater speed, we found no improvement in power output or completion time with deception of 5 % greater speed. Absence of improvements in power output was found despite a higher level of EMG activity in 5K_{105%} compared to 5K_{100%}. Specifically, the fall in power output despite high level of muscle activation about two kilometers after the start of exercise (Fig. 1) indicate that peripheral fatigue developed early during exercise and that muscle fatigue *per se* likely contributed to the subjects' inability to follow the virtual pacemaker in 5K_{105%}. Moreover, increases in perceived exertion and attentional focus on internal sources associated with the reduction in self-efficacy following the faster start in 5K_{105%} (Fig. 4) also suggest that the subjects' willingness to sustain a high level of muscle activation might have been impaired and could have also led to the progressive reduction in EMG amplitude and power output during the second part of the time-trial.

Our performance findings in 5K_{105%} are consistent with previous data showing no performance improvement with augmented deceptive feedback by the mean of false feedback showing speed to be 5% greater than actual speed (24). In contrast, Williams et al. (37), have recently reported similar improvements in completion time and mean power output during a 16.1 km cycling time-trial when participants were asked to compete against an onscreen avatar reproducing 102% or 105% of their fastest time-trial. No significant differences in RPE and self-efficacy were also found between the control and 105% condition in the aforementioned study. Together these results suggest that, in addition to differences in time-trial distance (16.1 km vs. 5 km), the way in which the role of the onscreen avatar was presented to the participants (competitor vs. pacemaker) might have stimulated different psychological processes and might explain, at least in part, discrepancies between studies.

What were the effects of augmented deceptive feedback on the recovery of the quadriceps muscle contractile function?

Whereas exercise-induced reduction in evoked potentiated twitch force were similar at the end of exercise between conditions, significant impairment in potentiated twitch force recovery was found in 5K_{102%}

and in 5K_{105%} (see Fig. 2). Specifically, when expressed as percent change from 5K_{100%} to 5K_{105%}, changes in potentiated twitch force were correlated to changes in power output and in completion time measured during the first half of the time-trial ($r > 0.76$, see Fig. 3). These correlations suggest that the impairment of quadriceps force recovery with augmented deceptive feedback might, at least in part, be accounted for by the effect of the higher power output and the faster start during the first part of time-trial. Moreover, the recovery of the potentiated twitch force was altered for at least 15 minutes following the end of exercise and this alteration was more pronounced at low (i.e. single and 10 Hz) compared to high (i.e. 100 Hz) frequency of stimulation (see Fig. 2). These findings suggest that this long-lasting force depression might be determined by alteration in intracellular Ca²⁺ concentration or myofibrillar Ca²⁺ sensitivity (1) in response to the faster pace and the presumably greater metabolic disturbance during the first half of the time-trial in 5K_{102%} and in 5K_{105%}.

Practical applications.

Our findings showed that performance improvement during deceptive time-trial is critically dependent on the magnitude of deception. Indeed, an improvement in time to complete the time-trial from 5K_{CTRL} and 5K_{100%} was only found with moderate magnitude of deception (i.e. 2% greater speed corresponding to 5% greater power output). The observed 2 % improvement in performance time (representative of 13 s) in 5K_{102%} is relatively large considering that the estimated worthwhile meaningful improvement in time to complete a cycling time-trial was ~0.7 % of the participant's fastest time-trial (representative of ~4 s) in the present study. We also showed that when the degree of deception was large (i.e. 5% greater speed), no performance improvement was found. Together our findings contribute to determine the boundaries within which the speed or the power output of a pacemaker can be increased (i.e. between 2 to 5 % of greater speed corresponding to 5 to 10% greater power output) to optimally enhance training stimulus or performance during cycling time-trials. Thus, this strategy of using deception can be finely tuned by coaches to aid athletes optimize performance.

CONCLUSION

Our findings showed that performance improvements during a cycling time-trial with moderate level of deception (i.e. 2% greater speed) are achieved with an increase in muscle activation during exercise and are associated with a greater impairment in voluntary activation and muscle contractile function following exercise. Specifically, the improved power output and the increase in metabolic demand with moderate level of deception (i.e. 2% greater speed) were accompanied by a significant alteration of the potentiated twitch force recovery. When the speed of the pacemaker was set too

fast (i.e. 5% greater speed) and the degree of deception therefore too large, no performance improvement was found.

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COMPETING INTEREST

The authors disclose any professional relationship with companies or manufacturers who will benefit from the results of the present study. The results of the present study do not constitute endorsement by ACSM. The results of the present study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

REFERENCES

- Allen DG, Lamb GD, Westerblad H. Skeletal muscle fatigue: cellular mechanisms. *Physiol Rev* 2008;88(1):287–332.
- Amann M. Central and peripheral fatigue: interaction during cycling exercise in humans. *Medicine and science in sports and exercise* 2011;43(11):2039–45.
- Amann M, Dempsey JA. Locomotor muscle fatigue modifies central motor drive in healthy humans and imposes a limitation to exercise performance. *J Physiol (Lond)* 2008;586(1):161–73.
- Amann M, Eldridge MW, Lovering AT, Stickland MK, Pegelow DF, Dempsey JA. Arterial oxygenation influences central motor output and exercise performance via effects on peripheral locomotor muscle fatigue in humans. *J Physiol (Lond)* 2006;575(Pt 3):937–52.
- Amann M, Proctor LT, Sebranek JJ, Pegelow DF, Dempsey JA. Opioid-mediated muscle afferents inhibit central motor drive and limit peripheral muscle fatigue development in humans. *J Physiol (Lond)* 2009;587(Pt 1):271–83.
- Blain GM, Mangum TS, Sidhu SK, et al. Group III/IV muscle afferents limit the intramuscular metabolic perturbation during whole body exercise in humans. *J Physiol (Lond)* 2016;594(18):5303–15.
- Borg E, Kaijser L. A comparison between three rating scales for perceived exertion and two different work tests. *Scand J Med Sci Sports* 2006;16(1):57–69.
- Burnley M, Vanhatalo A, Jones AM. Distinct profiles of neuromuscular fatigue during muscle contractions below and above the critical torque in humans. *J Appl Physiol* 2012;113(2):215–23.
- Cicchetti DV. Guidelines, criteria, and rules of thumb for evaluating normed and standardized assessment instruments in psychology. *Psychological Assessment* 1994;6(4):284–90.

AUTHOR CONTRIBUTIONS

G. P. D. and G. M. B. conceived the study, interpreted the data, and prepared the manuscript. G. P. D. analyzed the data. All authors executed the study and edited, revised, and approved the final version of the manuscript. All authors agree to be accountable for all aspects of the work, ensuring that questions related to the accuracy or integrity of any part are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

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- Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. Academic Press; 1977.
- Corbett J, Barwood MJ, Ouzounoglou A, Thelwell R, Dicks M. Influence of competition on performance and pacing during cycling exercise. *Medicine and science in sports and exercise* 2012;44(3):509–15.
- Gagnon P, Bussi eres JS, Ribeiro F, et al. Influences of spinal anesthesia on exercise tolerance in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 2012;186(7):606–15.
- Gagnon P, Saey D, Vivodtzev I, et al. Impact of preinduced quadriceps fatigue on exercise response in chronic obstructive pulmonary disease and healthy subjects. *J Appl Physiol* 2009;107(3):832–40.
- Gandevia SC. Spinal and supraspinal factors in human muscle fatigue. *Physiol Rev* 2001;81(4):1725–89.
- Hopkins WG. A new view of statistics [Internet]. *Sportscience* 2012; [cited 2016 Oct 13] Available from: <http://sports.org/resource/stats>
- Hopkins WG. Measures of reliability in sports medicine and science. *Sports Med* 2000;30(1):1–15.
- Hureau TJ, Ducrocq GP, Blain GM. Peripheral and Central Fatigue Development during All-Out Repeated Cycling Sprints. *Medicine and science in sports and exercise* 2016;48(3):391–401.
- Hureau TJ, Olivier N, Millet GY, Meste O, Blain GM. Exercise performance is regulated during repeated sprints to limit the development of peripheral fatigue beyond a critical threshold. *Exp Physiol* 2014;99(7):951–63.
- Keenan KG, Farina D, Maluf KS, Merletti R, Enoka RM. Influence of amplitude cancellation on the simulated surface electromyogram. *J Appl Physiol* 2005;98(1):120–31.
- Kufel TJ, Pineda LA, Mador MJ. Comparison of potentiated and unpotentiated twitches as an index of muscle fatigue. *Muscle Nerve* 2002;25(3):438–44.

21. Löscher WN, Cresswell AG, Thorstensson A. Central fatigue during a long-lasting submaximal contraction of the triceps surae. *Exp Brain Res* 1996;108(2):305–14.
22. Martin V, Millet GY, Martin A, Deley G, Lattier G. Assessment of low-frequency fatigue with two methods of electrical stimulation. *J Appl Physiol* 2004;97(5):1923–9.
23. Merton PA. Voluntary strength and fatigue. *J Physiol (Lond)* 1954;123(3):553–64.
24. Micklewright D, Papadopoulou E, Swart J, Noakes T. Previous experience influences pacing during 20 km time trial cycling. *Br J Sports Med* 2010;44(13):952–60.
25. Millar NC, Homsher E. The effect of phosphate and calcium on force generation in glycerinated rabbit skeletal muscle fibers. A steady-state and transient kinetic study. *J Biol Chem* 1990;265(33):20234–40.
26. Moritani T, Muro M, Nagata A. Intramuscular and surface electromyogram changes during muscle fatigue. *J Appl Physiol* 1986;60(4):1179–85.
27. Morton RH. Deception by manipulating the clock calibration influences cycle ergometer endurance time in males. *Journal of Science and Medicine in Sport* 2009;12(2):332–7.
28. Neyroud D, Maffiuletti NA, Kayser B, Place N. Mechanisms of fatigue and task failure induced by sustained submaximal contractions. *Medicine and science in sports and exercise* 2012;44(7):1243–51.
29. Pate E, Cooke R. Addition of phosphate to active muscle fibers probes actomyosin states within the powerstroke. *Pflugers Arch* 1989;414(1):73–81.
30. Rossman MJ, Garten RS, Venturelli M, Amann M, Richardson RS. The role of active muscle mass in determining the magnitude of peripheral fatigue during dynamic exercise. *Am J Physiol Regul Integr Comp Physiol* 2014;306(12):R934–40.
31. Schmidt NB, Lerew DR, Trakowski JH. Body vigilance in panic disorder: Evaluating attention to bodily perturbations. *Journal of Consulting and Clinical Psychology* 1997;65(2):214–20.
32. Sidhu SK, Hoffman BW, Cresswell AG, Carroll TJ. Corticospinal contributions to lower limb muscle activity during cycling in humans. *J Neurophysiol* 2012;107(1):306–14.
33. Stone MR, Thomas K, Wilkinson M, Jones AM, St Clair Gibson A, Thompson KG. Effects of deception on exercise performance: implications for determinants of fatigue in humans. *Medicine and science in sports and exercise* 2012;44(3):534–41.
34. Stone MR, Thomas K, Wilkinson M, St Clair Gibson A, Thompson KG. Consistency of perceptual and metabolic responses to a laboratory-based simulated 4,000-m cycling time trial. *Eur J Appl Physiol* 2011;111(8):1807–13.
35. Tabachnick BG, Fidell LS. *Using multivariate statistics, 5th*. Needham Height; 2007.1
36. Welch AS, Hulley A, Beauchamp M. Affect and self-efficacy responses during moderate-intensity exercise among low-active women: the effect of cognitive appraisal. *Journal of sport & exercise psychology* 2010;32:154–75.
37. Williams EL, Jones HS, Sparks SA, et al. Altered Psychological Responses to Different Magnitudes of Deception during Cycling. *Medicine and science in sports and exercise* 2015;47(11):2423–30.
38. Williams EL, Jones HS, Sparks SA, Marchant DC, Midgley AW, Mc Naughton LR. Competitor presence reduces internal attentional focus and improves 16.1km cycling time trial performance. *J Sci Med Sport* 2015;18(4):486–91.

