EFFECT OF FATIGUE ON MUSCLE COORDINATION IN REPEATED ALL-OUT BOUTS

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The aim of the present study was to analyse the fatigue occurring during repeated all-out short cycling bouts through mechanical and electromyographic (EMG) data and to focus on inter-muscle coordination. The results showed a significant decrease in peak power output without significant modifications in the EMG activity through sprint repetitions. However, the coordination timing between agonist and antagonist muscles was reduced. In conclusion, power output decreased during high-intensity repeated sprints due to the inability of quadriceps to maintain maximal force and owing to inter-muscle coordination limitations.

KEY WORDS: sprint, high-intensity, EMG, time delay, cycling

INTRODUCTION: A reduction in the force-generating capacity of muscles during intense dynamic exercises has been observed with unchanged or increased neural drive analysed from EMG data (Nummela, Vuorimaa, & Rusko, 1992; Kay, Marino, Cannon, St Clair Gibson, Lambert, & Noakes, 2001). The decrements of mechanical power that occur during high-intensity repeated short bouts are often explained by metabolic perturbations and skeletal muscle damages (Balsom, Seger, Sjödin, & Ekblom, 1992; Billaut, Giacomoni, & Falgairette, 2003; Gaitanos, Williams, Boobis, & Brooks, 1993). Nevertheless, when studying explosive movements, the inter-muscle coordination should also be taken into account to analyse performance evolution. It has been shown that the inter-muscle coordination pattern is modified during fatigue (Prilutsky, & Gregor, 2000). Recently, Neptune, & Kautz (2001) have highlighted the influence of activation and deactivation dynamics on optimal performance. This mechanism may play an important role in power output production during repeated sprints.
The aim of the present study was therefore to quantify the activation time delay adjustment between agonist and antagonist muscles during all-out short cycling bouts. We hypothesized that the activation time delay would be reduced with fatigue.

METHOD:
Subjects: Twelve well-trained subjects participated in the study (age 23 ± 2 years, mass 76.4 ± 4.2 kg, height 180.0 ± 5.5 cm, and physical activity 9.5 ± 4.6 h.wk-1). The experimental procedures were conducted in accordance with the local scientific committee. Each subject was fully informed about protocol modalities and provided written informed consent before all testing.
Equipment: All sprints were conducted on freewheel cycle ergometer (SRM, Jülich, Welldorf, Germany) equipped with toe-clips allowing subjects to wear cycling shoes. The torque applied on the crank was measured by a strain gauge located in the crank arm. The pedaling angular velocity and the torque were recorded online (200 Hz) from the ergocycle. The power output was calculated according to Jones, & Passfield (1998) formula, as follow:

$$\text{Power (W)} = \text{Torque (Nm)} \times \text{Angular Velocity (rad.s-1)}$$

Performance trial: After a standardized submaximal warm-up, subjects performed 10 repeated high-intensity 6-s sprints separated by 30-s rest. Starting pedal position was located 45° forward to the vertical axis and movement was initiated by the dominant leg. Subjects were instructed to perform each sprint as fast as possible and were strongly encouraged. They remained seated during the entire trial duration to prevent any alterations in the muscle recruitment pattern resulting from changes in posture. All bouts were performed at the same time of the day. Subjects were requested to refrain from heavy physical exercise two days prior to the trial and not to consume food two hours prior to the trial.

Data analysis: The peak power output (PPO) and the peak torque applied on crank (PT), defined as maximum values reached in 6 s were calculated during sprints 1, 5, and 10, and were used as fatigue indices.
EMG signals of vastus lateralis (VL), vastus medialis (VM), and biceps femoris (BF) muscles of the dominant leg were picked up via bipolar Ag-AgCl electrodes (Medicostest Blue Sensor R-00-S Electrode, Germany). Subjects wore a skin suit to prevent cable movement artifacts. Raw EMG signals were preamplified, filtered with a bandwidth frequency ranging from 1.5 to 500 Hz (common mode rejection ratio (CMRR) = 110 dB; Z input = 100 M ; gain = 1000), sampled at 1000 Hz, and digitized online by using a digital computer. During post-processing, EMG data of the 2nd, 3rd, and 4th pedal revolution (PR) of the sprints were analyzed. These EMG signals were full-wave rectified and the integral of the rectified EMG (IEMG) was calculated from a 25-ms sample of data. The same samples of data were further processed to assess the mean power frequency (MPF) by using a fast Fourier transformation.
For the activation time delay between agonist and antagonist muscles, a four order zero phase Butterworth low-pass filter (cutoff frequency: 8Hz) was used to eliminate cable movement artifacts. The muscle activation onset was then determined from EMG signal baseline using a constant electrical threshold over the time course of the sprints. For the three PR, the time between VL activation onset and BF activation onset and between VM activation onset and BF activation onset was calculated. All EMG parameters were analyzed with routines developed on MATLAB gait-analysis software (MATLAB, MathWorks Inc., Natick, MA, USA).
Statistics: A one-way analysis of variance (ANOVA) with repeated measures was used to detect differences in PPO and PT values. A two-way ANOVA with repeated measures was also used to detect differences in IEMG, MPF, and muscle activation time delay. Significant F-ratios were followed by post-hoc comparison using LSD Fisher's procedure. Significance was accepted at $P < 0.05$. Results were reported as mean ± SD.

RESULTS: The PPO and PT decreased significantly from sprint 1 to sprint 10 (-11% and -14.5%, respectively, $P < 0.01$). Similarly, the MPF of the EMG power spectrum was reduced ($P < 0.05$) except for the VM muscle (Table 1).

Table 1 Mean power frequency (MPF) of vastus lateralis (VL), vastus medialis (VM) and biceps femoris (BF) muscles for sprints 1, 5 and 10.

	Sprint 1	Sprint 5	Sprint 10
MPF - VL (Hz)	84.5 ± 16.6	78.4 ± 14.9	69.1 ± 12.9 **
MPF - VM (Hz)	80.3 ± 14.9	76.0 ± 15.1	71.7 ± 14.7
MPF - BF (Hz)	84.6 ± 21.1	80.7 ± 19.0	70.3 ± 17.9 *

*, ** significantly different from sprint 1 ($P < 0.05$ and $P < 0.01$, respectively).

An IEMG increase was observed among sprints without reaching significant values (+12.3% for VL, +9.2% for VM, and +17.8% for BF). Figure 1 displays the evolution of activation time delay between agonist and antagonist muscles. Among sprints, statistical analysis showed a significant time delay reduction between the onset of VL and VM activation and the onset of BF activation (sprint 1 vs sprint 10: -9.2ms, $P < 0.05$, Fig. 1a and sprint 1 vs sprint 10: -9.8ms, $P < 0.05$, Fig. 1b).

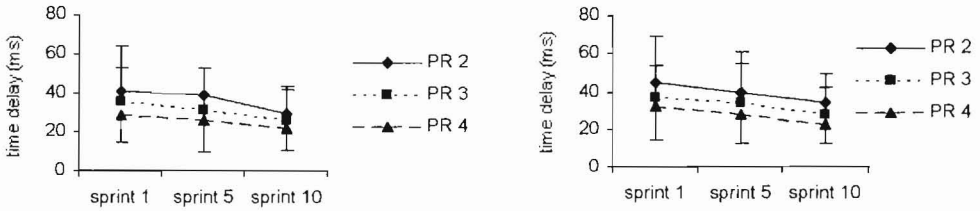

Figure 1: Timing of activation (time delay) between the onset of burst in VL and the onset of burst in BF (1a), and between the onset of burst in VM and the onset of burst in BF (1b) in sprints 1, 5 and 10, and for pedal revolutions 2, 3 and 4. * Significant difference from sprint 1, $P < 0.05$. § Significant difference from PR 2, $P < 0.05$.

DISCUSSION: The present study was designed to highlight the effect of fatigue on muscle coordination. The most interesting finding of this work was the muscle coordination alteration owing to fatigue. Indeed, the antagonist muscle (BF) activation was triggered earlier in the last sprint in parallel with force generation impairment.

The reduction in PPO and PT values in the last sprint were in line with results from studies using similar intermittent exercises (Balsom, Seger, Sjödin, & Ekblom, 1992; Billaut, Giacomoni, & Falgairette, 2003; Gaitanos, Williams, Boobis, & Brooks, 1993).

The EMG activity tended to increase but did not reach a significance level, suggesting that central neural drive to muscles was maintained (Taylor, Bronks, Smith, & Humphries, 1997). Thus, PPO and PT impairments would reflect substrate depletion, metabolic perturbations, impaired action potential propagation, excitation-contraction coupling failure, as well as an altered cross-bridges cycle activity (Gaitanos, Williams, Boobis, & Brooks, 1993; Häkkinen, & Komi, 1983; Taylor, Bronks, Smith, & Humphries, 1997). Force generation decrement might then result from fatigue occurring in the whole muscle as shown by the significant shift in EMG power spectrum toward lower frequencies.

The novelty of the present study was to investigate the fatigue occurring in repeated sprints throughout a simple method of muscle coordination timing analysis. According to Neptune, Kautz, & Hull (1997), the timing of muscle activation may reveal principles underlying muscular coordination. The present results indicated that the fatigue reduced the time delay between agonist and antagonist muscle activation (Fig. 1). In fact, the time delay between the VL and VM EMG onset and the BF EMG onset was reduced in the last sprint. This earlier BF muscle intervention suggests that activation and deactivation dynamics could have been affected by fatigue, and consequently could have modified muscle coordination. Thus, one can argued that the central nervous system was unable to adapt activation and deactivation pattern during repeated high-intensity 6-s sprints to avoid muscle coordination and performance decrements.

CONCLUSION: In the present study, in addition to the power output decrease due to a quadriceps force production collapse during high-intensity repeated cycling bouts, the neuromuscular system was unable to maintain an optimal coordination between agonist and antagonist muscles explaining in part the performance decrease.

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