Effects of fatigue on muscle activation during a 6s maximal sprint in cycling

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Summary

The aim of this study was to evaluate changes in muscle electromyography (EMG) activity in a maximal cycling sprint performed in fresh and fatigued condition. Sixteen competitive cyclists performed a 6 s seated maximal sprint at 90 rpm on an isokinetic ergometer, before and after a severe intensity trial to exhaustion (TTE) with EMGs recorded from major leg muscles. Peak power (Pmax) at 90rpm was reduced by 8.3±6.7% after TTE (p<0.001). Normalized EMG amplitude was lower for specific crank angles (p<0.05) in post- compared to pre-sprint. For gluteus maximus (GMAX), burst duration (p<0.048) decreased from pre- to post-sprint. We speculate that the lower activation of GMAX detected in the fatigued sprint could be partly responsible of the reduced capability of cyclists to express high power outputs after intensive training and competitions. This information can be used to evaluate specific training interventions to help to reduce this drop in muscle activation.

Introduction

In modern cycling, a rider's capacity to produce a maximal sprint in fatigued condition is one of the key determinants of elite performance [1]. In cycling, muscle fatigue is well reported to result in changes in EMG amplitude of some specific muscles as well as a prolonged period of muscle activity [2]. However, to date, how muscle activation changes during a short maximal sprint performed in fatigued conditions remains unclear. Therefore, the aim of this study was to evaluate differences in muscle EMG activity in a 6s maximal cycling sprint performed in fresh and fatigued conditions.

Methods

Sixteen healthy endurance competitive cyclists (8 males 36.0 ± 4.7 yrs, 73.0 ± 6.8 kg, VO_{2max} 57.0 ± 5.4 ml.kg^{-1}.min^{-1} and 8 females 30.0 ± 5.9 yrs, 56.9 ± 7.6 kg, VO_{2max} 52.2 ± 4.2 ml.kg^{-1}.min^{-1}), performed a 6 s seated maximal sprint at 90 rpm on an isokinetic ergometer, before and immediately after a TTE (587±154s) at 110% of critical power. During both sprints, EMG was recorded (2000 Hz) from Vastus Lateralis (VL), Rectus Femoris (RF) Vastus Medialis (VM), Tibialis Anterior (TA), Biceps Femoris (BF) Semitendinosus (ST) Gastrocnemius Lateralis (GL), Soleus (SOL) and GMAX muscles of the right leg. Pmax was calculated as the average of 3s power output after the participant reached 90rpm during the 6 s maximal sprint test both for unfatigued and fatigued conditions. The average EMG amplitude was calculated and burst duration was measured for each muscle for each pedal cycle and then averaged over 10 revolutions. The mean signals were then normalized to the peak EMG Root Mean Square (RMS) value from pre fatigue sprint. Statistical parametric mapping was used for time series variables, while paired t-tests was used for discrete variables.

Results and Discussion

After TTE, Pmax was reduced from sprint pre to sprint post (797.8±224 vs 726.3±196.8W respectively, p<0.002) by 8.3±6.7%. Normalized EMG amplitude was significantly reduced for specific regions of the crank cycle (p<0.05, Figure 1) in sprint post compared to sprint pre for VL (324-337°), RF (271-288°), VM (320-7°), TA (75-81°), BF (343-349°), ST (91-122°), GL (49-57°, 74-90° and 108-117°), SOL (269-282°) and GMAX (7-40°). Burst duration decreased from sprint pre to sprint post only for GMAX (18 ms, p<0.048).

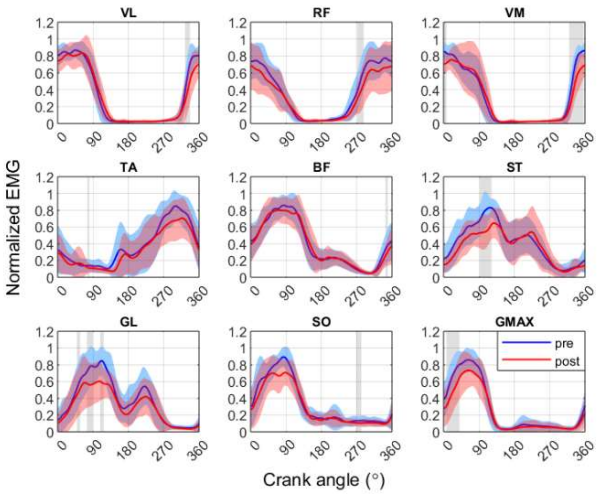

Figure 1: Group mean normalized EMG for each muscle in sprint pre and sprint post over a complete crank angle. Grey region denotes significant differences (p < 0.05).

Conclusions

According to Nielsen et al. [3], we speculate that the lower muscle activation detected in fatigue conditions for two of the main power producing muscles (VM, GMAX) could be one of main causes of the reduced capability of cyclists to express high power outputs after high intensity training or competitions. This information can be used to evaluate some specific training interventions that could help to reduce the observed drop in muscle activation.

References

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