Effect of including 30-s sprints in prolonged endurance exercise on muscular adaptations and gross efficiency in highly trained cyclists

Keywords: Cycling, sprint, mRNA, gross efficiency

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Introduction: Road cycling performance is mainly determined by maximal oxygen uptake (VO_{2max}), fractional utilization of VO_{2max} and gross efficiency (GE). However, performance is also determined by anaerobic capacity, especially in short and/or repeated sprints. A high volume of low-intensity endurance training (LIT) improves the ability to perform well in 4-6 h competitions in cyclists (Laursen, 2010, Schumacher and Mueller, 2002) and is shown to upregulate markers of mitochondrial biogenesis and angiogenesis (Skovgaard et al., 2016, Psilander et al., 2010). However, supplementation of training at higher intensities is needed to optimize both aerobic and anaerobic capacities. Training modalities such as repeated 30-s all-out sprints increases markers of mitochondrial biogenesis and angiogenesis (Taylor et al., 2016) but also markers of improved skeletal muscle ion-handling abilities (Iaia et al., 2008). These latter factors potentially explain the improved sprint performance seen after such training (Hostrup and Bangsbo, 2017). Interestingly, the combination of LIT and sprints induces superior increases in markers of mitochondrial biogenesis compared to LIT-only in trained subjects (Skovgaard et al., 2016). In the current study, our main aim was to investigate the effect of adding 30-s sprints to a long-lasting low-intensity ride on muscle signalling, GE, recovery of contractile function in highly trained cyclists. *Method:* 12 highly trained, male cyclists (VO_{2max}: 73.4 ± 4.0 $mL \bullet kg^{-1} \bullet min^{-1}$) completed a randomized, cross-over design containing a 4-h LIT session (50% of VO_{2max}) with 9 x 30-s sprints included (*E&S*) or a work-matched 4-h LIT session (*E*). GE, iEMG from m. Vastus lateralis (VL) and pedal force measurement were recorded in aerobic steady-state periods during exercise. Isokinetic leg extension was performed and muscle samples were collected from VL prior to exercise, as well as 20 min, 3 h and 24 h (isokinetic leg extension only) post exercise. *Results:* Expression of markers of mitochondrial biogenesis, PGC-1α splice 1, PDK4 and Tfam, increased ($P<0.05$) or tended to increase ($P<0.10$) in both conditions. The increase in PGC-1α splice 1 was smaller ($P<0.05$) after E&S than after E (Figure 1), whereas the increase in PDK4 was greater after E&S than after E. The angiogenic regulators HIF-α, VEGFA-stim and thrombospondin increased ($P<0.05$) to a similar extent in the two conditions. $Na^+/K^+\alpha 1$ mRNA expression was increased ($P<0.05$) after E only, with the relative change being different ($P<0.05$) from E&S. ClC1 and NHE1 mRNA expression was decreased ($P<0.05$) after E&S only, with the relative change being different ($P<0.05$) from E. GE was reduced ($P<0.05$) after the first set of sprints in E&S, with the relative change tending to be different from E ($P=0.06$). GE was reduced to the same extent after 4 hour of cycling in both conditions; 0.9±0.4 percentage points (PP) in E&S and 1.1±0.3 PP in E (Table 1; all $P<0.05$). Coherent with the decreased GE, the ventilation increased in an invert manner. Neither condition exhibited changes in RER, the angle of peak torque during the pedal stroke, pedalling cadence or iEMG. No correlation was seen between changes in either of these these variables and changes in GE. Peak torque in isokinetic leg extension was similar at 24 h post-exercise between conditions.

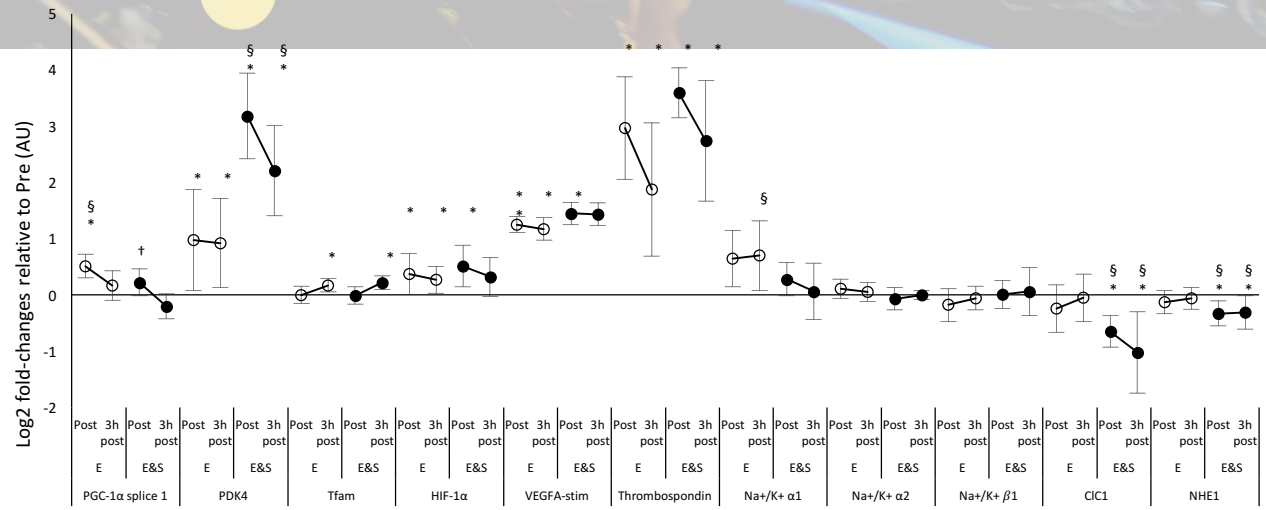

Figure 1. Log2 fold-changes in mRNA expression relative to pre exercise in Peroxisome proliferator-activated receptor gamma coactivator 1-alpha splice 1, pyruvate dehydrogenase kinase 4, Transcription factor A, Hypoxia-inducible factor 1-alpha, Vascular endothelial growth factor A, thrombospondin, Sodium-potassion pump subunits α1, α2, β1, chloride channel 1 and sodium-hydrogen antiporter. *: different (P<0.05) from pre. †: tendency to different (P<0.1) from pre, §: different (P<0.05) from change in E. Log2 fold-change in expression with 95% confidence intervals, *n*=12.

Table1: Gross efficiency, ventilation, respiratory exchange ratio, angle at which peak power is attained during a revolution, cadence and intramuscular EMG. *: different (P<0.05) from 5-10min. §: different (P<0.05) from E, Mean ± SD, n = 12.

		1st h		*2nd h*	*3rd h*	*4th h*	
		5-10 min	*30-35 min*	*30-35 min*	*30-35 min*	*30-35 min*	*58-60 min*
E&S	Gross efficiency (%)	19.7 ± 1.0	19.4 ± 0.7	19.1 ± 0.7 *§	19.1 ± 0.8 *	18.9 ± 0.9 *	18.8 ± 0.8 *
	V'E (L·min^{-1})	63.8 ± 1.9	65.3 ± 1.7 §	69.3 ± 1.9 *§	70.4 ± 1.8*§	70.3 ± 1.8 *§	69.8 ± 1.8 *§
	RER	0.89 ± 0.01	0.89 ± 0.01	0.89 ± 0.01	0.88 ± 0.01	0.89 ± 0.01	0.88 ± 0.01
	Angle at peak torque (degrees)	91.9 ± 1.2	92.1 ± 1.2	91.5 ± 1.2	91.9 ± 1.1	91.6 ± 1.0	91.3 ± 0.9
	Cadence (RPM)	86 ± 1	85 ± 2	86 ± 1	85 ± 1	86 ± 1	85 ± 2
	iEMG (mV)	80 ± 6	82 ± 7	87 ± 8	77 ± 10	82 ± 11	82 ± 10
E	Gross efficiency (%)	19.8 ± 0.7	19.7 ± 0.7	19.5 ± 0.8	19.3 ± 0.7	19.0 ± 0.8 *	18.8 ± 0.7 *
	V'E (L·min^{-1})	61.6 ± 1.8	63.0 ± 1.9	64.6 ± 1.8	64.9 ± 1.8	65.3 ± 1.8 *	65.9 ± 1.9 *
	RER	0.91 ± 0.01	0.89 ± 0.01	0.91 ± 0.01	0.89 ± 0.01	0.90 ± 0.01	0.90 ± 0.01
	Angle at peak torque(degrees)	93.1 ± 1.2	93.1 ± 1.1	92.7 ± 1.1	92.7 ± 1.0	93.1 ± 1.3	92.2 ± 1.3
	Cadence (RPM)	86 ± 1	86 ± 1	86 ± 1	86 ± 1	86 ± 1	86 ± 1
	iEMG (mV)	68 ± 4	74 ± 3	75 ± 5	77 ± 7	83 ± 2	88 ± 5

Conclusion: Implementing repeated 30-s sprint into a 4-h LIT session provides a different muscular stimulus than a work-matched LIT 4-h session in highly trained cyclists and differentially affected cellular systems for energy substrate regulation and muscle ion transport, including biomarkers for mitochondrial functions and angiogenesis. E&S seems to decrease mitochondrial biogenesis and shift towards more fatty acid metabolism. However, due to uncertainty in time course of activation and deactivation of these biomarkers, one should be careful about interpreting the acute signalling into chronic adaptations. Furthermore, GE decreased during prolonged exercise, and although the sprints seemed to negatively affect GE after the first series, the decreases in GE over the entire 4-h ride was similar across conditions. The recovery of contractile function was also similar 24 h post-exercise, indicating similar recovery rates across conditions.

Litterature: HOSTRUP & BANGSBO 2017. *J Physiol,* 595**,** 2897-2913. IAIA et al. 2008. *Am J Physiol Regul Integr Comp Physiol,* 294**,** R966-74. LAURSEN. 2010. *Scand J Med Sci Sports,* 20 Suppl 2**,** 1-10. PSILANDER et al. 2010. *Eur J Appl Physiol,* 110**,** 597-606. SKOVGAARD et al. 2016. *Physiol Rep,* 4. TAYLOR et al. 2016b. *Eur J Appl Physiol,* 116**,** 1445-54.