Effect of high-intensity, interval exercise on signalling proteins and gene expression in human skeletal muscle

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Short duration, high-intensity interval exercise is typically associated with strength or resistance training and linked to pathways that stimulate skeletal muscle growth. However, repeated sessions of such high-intensity, interval exercise induce rapid changes in skeletal muscle phenotype that are similar to those induced by traditional endurance exercise training, *i.e.* increased skeletal muscle oxidative capacity without marked hypertrophy (Burgomaster *et al.*, 2005). To investigate the effects of such high-intensity, interval exercise on signalling proteins and gene expression in human skeletal muscle, six untrained men (23 + 2 yrs) completed four 30s bouts of "all out" cycling exercise, separated by four min rest. Muscle (*v. lateralis*) samples were obtained by percutaneous needle biopsy before, after the first and fourth bouts and after three hours of passive recovery. Activation of signalling pathways was assessed by Western blotting using phosphospecific antibodies to AMP-activated protein kinase (AMPK), calcium/calmodulin-dependent protein kinase (CaMKII), p-38 mitogen-activated protein kinase (MAPK), protein kinase B (PKB) and 70-kDa S6 protein kinase (S6K). Skeletal muscle expression of selected genes (GLUT4, PGC1, PDK4 and COXIV) was assessed on muscle samples obtained before exercise and after three hours of recovery using real-time RT-PCR with specific primers and SYBR Green detection (BioRad). Phosphorylation (p-) of AMPK, CaMKII, p38 MAPK, PKB and S6K were unchanged after the first bout; however, p-AMPK and p-p38 MAPK were increased ($p < 0.05$) ~40% after the fourth bout, while p-PKB was reduced ($p < 0.05$) by ~50%. No changes in p-CaMKII, p-S6K or 4E binding protein phosphorylation were observed after exercise. PGC1 and PDK4 mRNA were increased ($p < 0.05$) ~3-fold after three hours of recovery, with no significant change in GLUT4 and COXIV mRNA. These data suggest that high-intensity, interval exercise activates signalling pathways known to contribute to mitochondrial biogenesis, with no activation of pathways responsible for growth-related protein synthesis. Such activation may explain the previously observed changes in muscle oxidative capacity and aerobic exercise performance following high-intensity, interval exercise training.

Burgomaster KA, Hughes SC, Heigenhauser GJF, Bradwell SN & Gibala MJ (2005) *Journal of Applied Physiology*, **98:** 1985-90.