Effect of exercise on Ca^{2+}-sensitive protein kinases in human skeletal muscle

A.J. Rose[1], B. Michell[2], B.E. Kemp[2] and M. Hargreaves[1], [1]Exercise, Muscle & Metabolism Unit, School of Health Sciences, Deakin University, Vic., 3125 and [2]St. Vincent's Institute of Medical Research, Fitzroy, Vic., 3065, Australia.

There is evidence in rodents that PKC (Richter *et al.*, 1989; Chen *et al.*, 2002) and CaMKII (Tavi *et al.*, 2003) activities are higher in contracting skeletal muscle, and that these kinases may regulate skeletal muscle function, including metabolism, during exercise. To investigate this in humans, healthy men (*n*=8, 24 ±5 yr, 23 ±2 kg•m^{-2}, $\dot{V}O_{2\ peak}$ = 51±6 ml•kg^{-1}•min^{-1}) performed cycle ergometer exercise for 40 min at 76±1 % $\dot{V}O_{2\ peak}$ with skeletal muscle samples taken at rest and after 5 and 40 min of exercise. PKC and CaMKII expression and activities were examined by immunoblotting and *in vitro* kinase assays. There were no differences in maximal (+Ca^{2+}/CaM) CaMKII activity during exercise compared with basal. Autonomous (−Ca^{2+}/CaM) CaMKII activity was 9 ±1% of maximal at rest, unchanged at 5 min, and increased to 17 ±1% (P<0.01) at 40 min. There were no differences in CaMKII expression (P>0.1). There were no changes in cPKC or PKCθ activities (P>0.1), however aPKC activity was ~70% higher (P<0.05) at 5 and 40 min and total PKC activity was slightly higher at 40 min in an enriched membrane fraction (P<0.05).

The activities of these kinases were also examined in response to maximal aerobic exercise. Healthy men (*n*=9, 25 ±5 yr, 24 ±2 kg•m^{-2}, 52 ±9 ml•kg^{-1}•min^{-1}) performed cycle ergometer exercise for 10 min at 50 %$\dot{V}O_{2\ peak}$, after which the workload was increased to elicit 100 %$\dot{V}O_{2\ peak}$ with muscle samples taken at rest and at volitional fatigue. Autonomous CaMKII activity was increased by 74 ±17% (P<0.001) with no change in maximal CaMKII activity. There were no changes in total PKC, PKCδ, PKCθ, or aPKC activities.

These data demonstrate that CaMKII and aPKC are activated in contracting skeletal muscle, and thus may represent key signalling proteins potentially regulating skeletal muscle function and metabolism during exercise in humans.

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