High-intensity interval training increases the rate of mitochondrial fatty acid oxidation in rat skeletal muscle

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Purpose: High-intensity interval training (HIIT) can increase mitochondrial volume and density and induce an increase in oxidative capacity in skeletal muscle. HIIT also increases substrate transport protein associated with carbohydrate such as glucose and lactate. However, it is unclear whether HIIT alters the intrinsic capacity of mitochondria to oxidize substrates and fatty acid transport protein in skeletal muscle. It is known that there are subsarcolemmal (SS) and intermyofibrillar (IMF) mitochondria in skeletal muscle. We hypothesized that HIIT would increase rates of fatty acid and pyruvate oxidation in IMF mitochondria, because IMF mitochondria distribute between the myofibrils and we predicted that exercise training would stimulate IMF mitochondria to a greater extent. To clarify this hypothesis, we examined effects of HIIT on contents of mitochondrial substrate transporter protein, mitochondrial enzyme activities and substrates oxidation rates in isolated SS and IMF mitochondria in skeletal muscle.

Methods: Sprague–Dawley male rats were divided into control or HIIT groups, and the HIIT group performed 10 bouts of 1 min high-intensity treadmill running (30–55 m/min) with 2 min rest 5 days/wk for 4 wk. Thereafter hindlimb muscles were harvested 48 hr after last bout of training. The analyses were carried out at whole muscle level or isolated mitochondrial level.

Results: HIIT increased substrates transporters, namely fatty acid translocase (FAT)/CD36, glucose transporter 4 (GLUT4) and monocarboxylate transporter (MCT) 1, in whole muscle. Also mitochondrial markers, citrate synthase activity and cytochrome c oxidase subunit IV protein, were increased with HIIT. These results suggest that the capacity of substrate transport and mitochondrial density were improved in whole muscle. In isolated mitochondria, HIIT increased the rate of fatty acid oxidation in both SS and IMF mitochondria in red muscle and the increase in mitochondrial palmitate oxidation was accompanied with concomitant increase in β-hydroxyacyl-CoA dehydrogenase activity. However, pyruvate oxidation was not altered in SS and IMF mitochondria in red and white muscle with HIIT. Similarly, MCT2, a pyruvate transporter, and pyruvate dehydrogenase E1α protein contents per isolated mitochondrial protein remained unchanged after HIIT.

Conclusions: These results suggest that high-intensity interval training improved fatty acid metabolism due to enhanced fatty acid transport protein, increased mitochondrial content in whole muscle, and to the increased intrinsic capacity for fatty acid oxidation in isolated SS and IMF mitochondria.