



## Exercise Biochemistry Review

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### Effect of exercise training on mitochondrial content after ischemia in the cerebral cortex of rat

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**Objective** To investigate the effect exercise training on mitochondrial content after ischemia in the cerebral cortex, and the balance between mitochondrial biogenesis and mitophagy in this process.

**Methods** Male Sprague-Dawley rats were randomly divided into 4 groups ( $n = 8$ ): Sham operation group (SH), Sham operation and training group (ST), ischemia control group (IC), ischemia and training group (IT). The ischemia model rats were subjected to right middle cerebral artery occlusion which was produced by the intraluminal suture technique. The rats in the sham group underwent the same procedure except for the occlusion of the middle cerebral artery. Fourteen days after operation, the exercise training animals were exercised on a motor-driven rodent treadmill at a speed of 10 m/min, 5% grade for 30 min/day, 5 days/week for 4 weeks. Mitochondrial membrane potential was determined using JC-1. ATP synthesis capacity was determined using a bioluminescence technique. The protein expression of VDAC-1, COXIV, PGC-1 $\alpha$ , Tfam, PINK1 and Parkin in cerebral cortex were detected by Western-blotting.

**Results** Compared with SH group, ischemia attenuated mitochondrial membrane potential, ATP synthesis capacity, and the expression of VDAC-1, COXIV, PGC-1 $\alpha$ , Tfam in cerebral cortex ( $P < 0.05 \sim 0.01$ ). Furthermore, ischemia increased the expression of PINK1 and Parkin ( $P < 0.05 \sim 0.01$ ). Compared with IC group, exercise training elevated mitochondrial membrane potential, ATP synthesis capacity, and the expression of VDAC-1, COXIV, PGC-1 $\alpha$ , Tfam, PINK1, Parkin in IT group ( $P < 0.05 \sim 0.01$ ).

**Conclusions** A combination of reduced mitochondrial biogenesis and increased mitophagy seems to be responsible for the decrease in mitochondrial content after ischemia. Exercise training after ischemia elevated mitochondrial content and function in cerebral cortex, which may be mediated by appropriately increase and co-regulation of mitochondrial biogenesis and mitophagy.