



## Exercise Biochemistry Review

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### Effect of Aerobic Exercise on Myocardial Mitochondria Fusion-Fission in Chronic Intermittent Hypoxia Mice

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**Objective** Obstructive sleep apnea hypopnea syndrome (OSAHS) could impair the function of multiple organs because of repeated chronic intermittent hypoxia (CIH)/reoxygenation during sleep. Which is harmful to the health and economy of patients. It is found that exercise can protect the function of multiple organs in OSAHS patients, especially the heart function, while the specific mechanism is not clear. The purpose of this paper is to investigate the effects of aerobic exercise on cardiac contractile function and mitochondrial fusion-division of CIH mice.

**Methods** C57/BL6 mice of 6 weeks old were used in this experiment. The mice were divided into 3 groups randomly: control group (C), intermittent hypoxia group (CIH), intermittent hypoxia plus exercise group (CIH+E), 10 mice in each group. A self-designed computer controlled chronic intermittent hypoxia device was used. Group C was treated with normal feeding for 5 weeks. The CIH group was treated with intermittent hypoxia for 8 hours a day for 5 weeks. The CIH+E group were treated with suitability running on treadmill after CIH treatment at the first week. Starting from week 2, this group were treated with 60 minutes of running stage after 8 hours of CIH treatment, total 5 weeks.

After the experiment, the cardiac function of each group was measured by two-dimensional echocardiography. The interventricular septal thickness (IVS), left ventricular posterior wall thickness (LVPW), ejection fraction (EF) were measured. Ultrastructure and morphology of mitochondria in cardiomyocytes were observed by transmission electron microscopy (TEM). The expression of mitochondrial fusion (Mitofusin 1 (Mfn1), Mitofusin 2 (Mfn2), Opa-ticatrophylopa (Opa1)) and fission (dynamic in-related protein 1 (Drp1), mitogen 1, Fission1 (Fis1)) related genes were detected by RT-PCR.

**Results** Intermittent hypoxia reduced IVS and EF in mice, suggesting that CIH caused the loss of cardiac myocytes. Compared with C group, LVPWd+IVSd in CIH+E group was significantly lower than that in C group ( $P < 0.01$ ) and EF was significantly lower than that in CIH E group. The cardiomyocyte loss and cardiac contractile function of CIH+E mice were improved by aerobic exercise for 4 weeks. LVPWd+IVSd in CIH+E group was better than that in CIH group. The EF value of ( $P < 0.01$ ) in group A was significantly higher than that in group CIH ( $P < 0.01$ ). Intermittent hypoxia decreased heart weight/body weight ratio (HW/BW) and left ventricular weight/tibia length left ventricular weight/tibia (LVW/TL). Compared with the C group, the HW/BW in the CIH group was significantly lower than that in the C group, and the HW/BW in the CIH group was significantly higher than that in the CIH group. LVW/TL also increased significantly ( $P < 0.05$ ), suggesting that intermittent hypoxia could lead to necrosis and decrease in number and weight of myocardial cells. (2) The results of mitochondrial electron microscope showed that compared with group C, the number of mitochondria decreased, the structure of mitochondria changed, and the number of myocardial mitochondria in CIH+E group increased after 4 weeks of aerobic exercise. Mitochondrial membrane and mitochondrial crest were repaired to improve the structure of myocardial mitochondria. (3) compared with C group, the mitochondrial fusion related gene Mfn2/OPA1 in CIH group was significantly lower than that in C group ( $P < 0.05$ ,  $P < 0.05$ ). Aerobic exercise significantly increased Mfn1, mfn2, OPA1 in CIH+E group than that in CIH group. Compared with C group, Mfn2, OPA1 in CIH group was significantly higher than that in CIH group. The Drp1 of the CIH+E group was significantly

lower than that of the CIH group, and the Drp1 of the CIH+E group was significantly lower than that of the CIH group. 1 compared with CIH group,  $P < 0.01$  was significantly lower than that of CIH group. **Conclusions** (1) aerobic exercise has protective effect on cardiac contractile function in intermittent hypoxia mice. (2) aerobic exercise can promote mitochondrial fusion in CIH mice, inhibit mitochondrial division, and protect myocardium by mediating mitochondrial function.