Cardiovascular Rehabilitation and Sports Medicine

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Differential effects of exercise training on K^+-mediated vasodilation of deep femoral artery and cerebral arteries of rat and the underlying electrophysiological changes

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Objectives: A moderate increase in extracellular $[K^+]$ ($[K^+]_e$) from 4 to 8 mM induces relaxation of small arteries and arterioles, which is known to be mediated by increased slope conductance of inwardly rectifying K^+ (Kir) channels and partly by Na^+/K^+-ATPase. The K^+-vasodilation is an important mechanism for exercise-induced hyperemia in skeletal muscle.
Methods: We investigated whether I_{Kir} and K^+-vasodilation in deep femoral arteries (DFA, inner diameters around 0.2 mm) using Patch-clamp technique and video-analysis of pressurized arteries in control and exercise-trained rats (ET-rats) that went through 3 times of treadmill-running (20 m/min, 30 min, six days for two week). The effects of ET on K^+-vasodilations and IKir were also compared with cerebral arteries and mesenteric arteries.
Results: The K^+-vasodilation of DFA and the density of I_{Kir} and voltage-gated K^+ current (I_{Kv}) were increased in ET-rats. The myogenic tone of DFA was unchanged in ET-rats. Although the functional up-regulations of I_{Kir} and I_{Kv} were also observed in cerebral arteries, the K^+-vasodilation was not increased in ET-rats. Interestingly, background Na^+ conductance was also increased in the cerebral arterial myocytes while not in DFA myocytes from ET-rats.
Conclusions: We firstly report that regular exercise up-regulates I_{Kir} in the myocytes of DFA and cerebral artery. Albeit the common increase of I_{Kir}, augmentation of K^+-relaxation was observed in DFA only, which might be due to the increased Na^+ conductance in cerebral artery of ET-rats. The increases of I_{Kir} and K^+-vasodilation of skeletal arteries suggest novel mechanisms of improved exercise hyperemia by physical training.

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High versus moderate-intensity aerobic exercise effects on apoptosis, oxidative stress and metabolism of infracted myocardium in rat model

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Objectives: The aim of this study was to investigate and compare the effects of continuous moderate-intensity training (CMT) and high-intensity interval training (HIT) on cardiac function recovery and potential mechanism associated with mitochondria function including apoptosis, oxidative stress and glucolipid metabolism.
Methods: 8 week wistar rats underwent MI or sham surgery. Rats in the training groups were submitted to a continuous running training (MI+CMT) or interval running exercise (MI+HIT) on a treadmill for 8 weeks while rats in the sham and MI group almost kept sedentary in the same period. Before and after the exercise training, echocardiographic parameters and exercise capacity were measured. Western blot was used to measure the levels of apoptosis protein bax, bcl-2 and caspase-3 and signal pathway protein PI3K, Akt, p38MAPK and AMPK. Concentrations of biomarkers of oxidative stress such as MDA, SOD, and GPx were determined by ELISA assay. mRNA level and activity of the key enzymes for glycolysis and fatty acid oxidation, carnitine palmitoyltransferase-1 (CPT-1) and phosphofructokinase-1 (PFK-1), as well as the rate of ATP synthesis were also measured.
Results: Compared with the rest MI group, exercise capacity and cardiac function were significantly improved after aerobic exercise training (AET), especially after HIT. LVEF and FS were further improved in the MI-HIT group than the MI-CMT group [LVEF(%): 72.7±1.23 vs. 64.2±1.07, P<0.05; FS(%): 36.4±1.32 vs. 32.1±1.53, P<0.05].Two forms of AET almost equally attenuated apoptosis of infarcted myocardium with increased protein expression of bax and decreased bcl-2 and caspase-3. CMT and HIT both alleviated oxidative stress by decreasing the concentration of MDA and increasing the concentration of SOD and GPx. Meanwhile, CMT and HIT all significantly increase the level of mRNA and activity of PFK-1 and CPT-1as well as the ratio of ATP synthesis compared with the rest MI group, but HIT could further improve the mRNA level of PFK-1, activity of PFK-1 and CPT-1, as well as ATP synthesis than that of CMT.
Conclusions: Our study demonstrated that HIT was superior to CMT as to improving exercise capability and cardiac function in myocardial infarcted rat model associated with alleviating oxidative stress, ameliorating glucolipid metabolism and boosting ATP production.

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Role of Endothelial Progenitor Cells and Nitric Oxide in Collateral Circulation Formation in Ischemic Myocardium Induced by Physiological Ischemic Training

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Objectives: To evaluate changes in endothelial progenitor cells (EPCs) and Nitric oxide (NO) in rabbits with ischemic myocardium in association with collateral circulation formation induced by physiological ischemic training (PIT).
Methods: Controlled myocardial ischemia was modelled by a water balloon constrictor implanted on the left ventricular branch in a rabbit. The models were assigned randomly into six groups: sham-operation group (SO); limb ischemic training group(LT) was induced by three cycles of 3-minute ischemic followed by 5 minutes of reperfusion using tourniquets on the hind limbs for 4 weeks; myocardial ischemic training group (MT) was subjected to two cycles of 2-minute myocardium ischemic followed by 10minutes of reperfusion by deflation and inflation of the water balloon for 4 weeks; physiological ischemic training group (PT) was induced by two cycles of myocardium ischemic training as MT plus three cycles of limb ischemic training as LT; PT with pretreatment with the EPCs promoter (ProEPCs-PT) and PT with pretreatment with the EPCs inhibitor (InhiEPCs-PT). At the end point, we used immunohistochemistry, nitrate reductase method, fluorescence- activated cell sorter to assess collateral capillary density (CD), NO and EPCs in regional ischemic myocardium and peripheral circulation.
Results: The EPCs count was significantly higher in the PT than in the SO, LT and MT. PT has more EPCs than InhiEPCs-PT ($P<0.05$), but less than Pro-EPCs-PT ($P<0.05$).Compared with the other three training groups (SO, LT, MT), PT had the highest CD ($P<0.05$) and NO level ($P<0.05$), the effect was enhanced by EPCs promoter ($P<0.05$), but restrained by EPCs inhibitor ($P<0.05$), while results between SO and LT had no difference. CD and NO level in ischemic myocardium were highly correlated with the number of EPC count (r=0.780; r=0.699).
Conclusions: Physiological ischemic training may promote the quantity and homing to the ischemic myocardium of EPCs, and enhance secretion of NO, resulting in an increase in angiogenesis in remote ischemic myocardium.

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Six weeks physical exercise improve obesity-associated hyperactivation of renin-angiotensin system in obese children

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Objectives: In recently years, rising prevalence of childhood and adolescents obesity is one of the major problems in both developed and developing countries, which confers an increasing risk for future development of type 2 diabetes and cardiovascular disease. The renin-angiotensin-aldosterone system has been causally implicated in obesity-associated endothelial dysfunction and insulin resistance. Recently studies suggest that renin-angiotensin system in the activated state for a long time in obese children, which may induce many cardiovascular risk factors. Although physical exercise has been shown to decrease cardiovascular disease risk factors and improve vascular function and insulin sensitivity in obese individuals even in the absence of weight loss. However, it is unclear whether exercise reduces activated rennin-angiotensin system in obese children and adolescent. We studied the effect of six physical exercise intervention on the circulating renin-angiotensin-aldosterone system, endothelial function and insulin sensitivity in obese children.
Methods: 14 obese children (8 boys and 6 girls), ranging from 10 to 14 years of age, were recruited at closed weight loss camp. Obesity was defined according to the international age-related cut-off points for childhood obesity. 8 lean control subjects were also recruited to allow cross-sectional comparison, were studied only at baseline. All obese subjects participated in an exercise training program at a closed weight loss camp for 6 weeks. The aerobic exercise program include basketball, swimming, jogging and other games, which was maintained at 60% to 70% of maximum HR for about 40min, and was performed 5 sessions a week for six weeks. All participants' blood samples were analyzed for angiotensin II and aldosterone at baseline and at after 6 weeks exercise protocol.
Results: Obese subjects had significantly higher angiotensin II (88.27±12.34 pg/ml vs 73.74 ±13.28 pg/ml, P<0.01) and aldosterone (96.51±17.45 pg/ml vs 75.65±15.59 pg/ml, P<0.01) levels compare to control subjects. Results shows the data after 6 weeks exercise intervention compared to before exercise intervention in obese subjects. The angiotensin II (88.27±12.34 pg/ml vs 78.72±14.82 pg/ml, P<0.05) and aldosterone (96.51±17.45 pg/ml vs 84.28 ±18.24 pg/ml, P<0.05) levels were significantly decreased after 6 weeks exercise intervention as compared to before exercise intervention.
Conclusions: Obese children had higher circulating angiotensin II and aldosterone than control children. Six weeks physical exercise lowers circulating angiotensin II and aldosterone levels in obese children; it is suggest that the renin-angiotensin system were decreased in obese children by exercise, which may contribute to the reduced cardiovascular disease risk factors due to cardiovascular risk factors are associated with activation of the tissue rennin-angiotensin system, especially for hypertension, insulin resistance, vascular endothelial dysfunction.