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Astaxanthin Reduces High Intensity Training Induced Myocardial Cell Apoptosis Via Activating Nrf2 in Rats

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Objective Long-term intensive training may led to ischemia oxygen reaction and increase the ROS. Astaxanthin, as the super antioxidant, was investigated to against anti-oxidative stress. By supplementing the astaxanthin, we wanted to observe if it can mediated Nrf2 reduces myocardial cell oxidative injury in rats after high intensity training of 6 weeks.
Methods 7-week SD male rats were divided into 3 groups randomly: control group (C group， n =10) ， high intensity training group (HT group， n = 15)， astaxanthin and high intensity training group (HTA group， n = 15) . The rats in HTA group were given with astaxanthin 20 mg /kg·d and in HT group were given with oil during the training day． The serum cTnI， myocardial apoptosis index, the expression of myocardial BAX, Bcl2, Nrf2, HO-1, myocardial MDA， SOD and T- AOC activity were measured 24 hours after the last training.
Results After 6-week tranning of high intensity, compared with group C, the serum cTNI, myocardial apoptosis index, the expression of BAX and myocardial MDA were significantly higher in group HT($P<0.01$).The Bcl2/Bax, the expression of HO-1, SOD and T-AOC activity were significantly declined ($P<0.01$). After the intervention of 6-week astaxanthin, compared with group HT, the serum cTNI, myocardial MDA, the myocardial apoptosis index, the expression of BAX were significantly lower in HTA group (cTNI(ng/ml): 1.16±0.27 VS 2.47±0.39, $P<0.05$; myocardial apoptosis index: 164.27±3.98 VS 196.20±9.65, $P<0.01$; BAX: 58.40±5.95 VS 78.03±3.80, $P<0.01$). Finally, Bcl2/Bax, SOD, T-AOC activity, the expression of Nrf2 and HO-1 were significantly higher (Bcl2/Bax : 1.92±0.10 VS 1.19±0.18, $P<0.01$; SOD(U/mg): 52.38±6.15 VS 38.32±3.36, $P<0.01$; T-AOC(U/mg): 30.22±4.07 VS 23.76±3.20, $P<0.01$; Nrf2: 93.61±8.53 VS 74.26±6.69, $P<0.01$; HO-1: 84.99±13.78 VS 64.22±11.39, $P<0.05$).
Conclusions The supplement of astaxanthin can mediate Nrf2 signaling pathway, and elevate the expression of Nrf2 and HO-1. Then it can increase the activity of SOD and T-AOC and reduce the myocardial oxidative level and myocardial apoptosis in rats caused by 6-week high intensity training. Finally, the structure and function of heart tissue are back to normal.