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摘要:

摘要目的: 观察游泳训练对ApoE基因敲除小鼠胰岛素抵抗模型血清游离脂肪酸(FFA)、肝脏组织过氧化物酶体增殖物激活受体- γ (PPAR- γ)及肉碱棕榈酰转移酶-1(CPT-1)、酰基辅酶A脱氢酶(MCAD)mRNA表达的影响, 初步探讨游泳训练改善ApoE基因敲除小鼠胰岛素抵抗(IR)的可能机制。**方法:** 选取8周雄性ApoE基因敲除小鼠26只, 随机分为: 高脂运动组(n=13)和高脂静止组(n=13)。高脂运动组小鼠给予高脂饮食加游泳训练12周, 高脂静止组除不进行游泳训练外, 余同高脂运动组。另以健康雄性C57BL/6J(n=10)小鼠为正常对照组, 饲料喂养12周。干预12周后, 测各组小鼠空腹胰岛素(FIN)、空腹血糖(FPG), 并以HOMA法计算胰岛素抵抗指数(IRI), 确定胰岛素抵抗模型建立; 全自动生化分析仪测定血清中总胆固醇(TC)、甘油三酯(TG)、高密度胆固醇脂蛋白(HDL)、低密度胆固醇脂蛋白(LDL)、游离脂肪酸(FFA)含量; RT-PCR法测肝脏组织中PPAR- γ 、CPT-1、MCAD mRNA表达水平。结果: 运动干预12周以后: ①与正常对照组相比较, 高脂静止组体重显著增加($P<0.05$); 与高脂静止组比较, 高脂运动组体重显著下降($P<0.05$)。②与正常对照组相比较, 高脂静止组FPG、Homa-IRI水平明显升高(P 均 <0.01); 与高脂静止组相比较, 高脂运动组FIN、FPG、Homa-IRI明显降低(P 分别 <0.05 、 0.01 、 0.01)。③与正常对照组相比较, 高脂静止组TC、LDL、FFA水平明显升高(P 均 <0.01); 与高脂静止组相比较, 高脂运动组TC、LDL、FFA水平明显降低(P 分别 <0.05 、 0.05 、 0.01), HDL水平明显升高($P<0.05$)。④与正常对照组相比较, 高脂静止组PPAR- γ 、CPT-1、MCAD mRNA表达明显降低(P 均 <0.01); 与高脂静止组相比较, 高脂运动组PPAR- γ 、CPT-1、MCAD mRNA表达明显增加(P 均 <0.01)。结论: 游泳训练可上调肝脏组织PPAR- γ 表达, 进而上调CPT-1、MCAD的表达, 改善小鼠脂代谢, 从而改善ApoE基因敲除小鼠胰岛素抵抗。

关键词: [胰岛素抵抗](#) [运动](#) [过氧化物酶体增殖物激活受体- \$\gamma\$](#) [肉碱棕榈酰转移酶-1](#) [中链酰基辅酶A脱氢酶](#)

The effect of swimming exercise on the expression of peroxisome proliferator-activated receptor- γ and carnitine palmitoyl transferase-1, medium-chain acyl-coenzyme A dehydrogenase of lipid metabolism in the apolipoprotein E knockout mice [Download Fulltext](#)

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Fund Project:

Abstract:

Abstract Objective: To observe the effects of swimming training on serum free fatty acid (FFA) level and the expressions of peroxisome proliferator-activated receptor- γ (PPAR- γ), carnitine palmitoyl transferase-1(CPT-1), medium-chain acyl-coenzyme A dehydrogenase(MCAD) mRNA in the apolipoprotein E (ApoE) knockout mice and to explore the possible mechanisms of swimming training in improving insulin resistance (IR) of ApoE-null mice. **Method:** Twenty-six male ApoE knockout mice randomly divided into two groups: the high-fat diet group (HFD, n=13) and the high-fat diet group with exercise training (HFD+Ex, n=13). The mice of HFD+Ex group were fed with high-fat diet with exercise training for 12 weeks. The intervention of HFD group was identical to that of HFD+Ex group except swimming training. healthy male C57BL/6J (ND, n=10) mice, as the control group, were fed with normal diet (ND) for 12 weeks. After 12 weeks, the mice were dissected, and the liver tissues were excised off immediately. Serum insulin and glucose were determined, while Homa-IRI was calculated to ascertain the establishment of IR. Serum total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), FFA were determined. The PPAR- γ , CPT-1, MCAD mRNA in liver were detected by reverse transcription polymerase chain reaction (RT-PCR). **Result:** ①Compared with ND group, the body weight of mice of HFD group was significantly higher ($P<0.05$). Compared with HFD group, the body weight of mice of the HFD+Ex group was significantly lower ($P<0.05$). ②Compared with ND group, fasting glucose, insulin and Homa-IRI of HFD group were significantly higher ($P<0.01$ all). Compared with HFD group, fasting insulin, glucose and Homa-IRI of HFD+Ex group was significantly lower ($P<0.05$, 0.01 , 0.01 separately). ③Compared with ND group, TC, LDL, FFA of HFD group were significantly higher ($P<0.01$ all). Compared with HFD group, TC, LDL, FFA of HFD+Ex group were significantly lower ($P<0.05$, 0.05 , 0.01 separately), HDL was significantly higher ($P<0.05$). ④Compared with ND group, the expressions of PPAR- γ , CPT-1, MCAD mRNA of HFD group decreased significantly ($P<0.01$ all). Compared with HFD group, the expressions of PPAR- γ , CPT-1, MCAD mRNA increased significantly ($P<0.01$ all). **Conclusion:** Swimming training can improve IR of ApoE-null mice which possibly acts by upregulating the expressions of PPAR- γ , CPT-1 and MCAD mRNA.

Keywords: [insulin resistance](#) [exercise](#) [peroxisome proliferators-activated receptor gamma](#) [carnitine palmitoyl transferase 1](#) [medium chain acyl-coenzyme A dehydrogenase](#)