

论著

梓醇对鱼藤酮致小鼠中脑和纹状体线粒体酶活性改变的改善作用

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摘要 目的 研究梓醇对鱼藤酮致小鼠脑组织线粒体酶活性的影响。方法 昆明小鼠分别ip给予梓醇10 mg·kg⁻¹, 连续7 d后, 改ip给予鱼藤酮1.5 mg·kg⁻¹, 连续28 d; 或ip给予鱼藤酮1.5 mg·kg⁻¹, 连续28 d后, 改ip给予梓醇10 mg·kg⁻¹, 连续7 d。取小鼠中脑和纹状体提取线粒体测定线粒体复合物酶 I、II、II+III、IV、线粒体一氧化氮合酶(mtNOS)活性、线粒体膜电位以及线粒体内活性氧(ROS)。结果 与正常对照组相比, 鱼藤酮组中脑和纹状体线粒体复合物 I 和IV活性明显下降($P<0.01$), 预防或治疗性地给予梓醇后活性明显上升($P<0.05$); 线粒体复合物 II 和 II+III活性各组间无明显差异。与正常组相比, 鱼藤酮组小鼠中脑和纹状体线粒体膜电位下降($P<0.01$), mtNOS活性升高($P<0.05$), ROS的生成增多($P<0.05$), 预防或治疗性地给予梓醇后中脑和纹状体线粒体膜电位升高($P<0.01$), mtNOS活性降低($P<0.05$), ROS的生成减少($P<0.05$)。结论 梓醇可能通过恢复脑内线粒体复合物酶活性和膜电位水平、减少线粒体内ROS生成的作用而抑制鱼藤酮诱导的脑损伤。

关键词 [梓醇](#) [鱼藤酮](#) [线粒体](#)

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Improving effect of catalpol on changes of mitochondrial respiratory chain activity in mice induced by rotenone

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Abstract

OBJECTIVE To investigate the effect of catalpol on the activities of mitochondrial enzyme complex in mouse brain induced by rotenone. **METHODS** Kunming mice were intraperitoneally injected with catalpol 10 mg·kg⁻¹ for 7 d and then with rotenone 1.5 mg·kg⁻¹ for 28 d, or the mice were intraperitoneally injected with rotenone 1.5 mg·kg⁻¹ for 28 d and then with catalpol 10 mg·kg⁻¹ for 7 d. The striatum and mesencephalon were isolated and the activity of mitochondrial enzyme complexes I, II, II+III, IV, mitochondrial nitric oxide synthase (mtNOS), mitochondrial membrane potential and reactive oxygen species (ROS) in the brain of mice were measured, respectively. **RESULTS** Compared with the normal group, the activity of complex I, IV in the brain was significantly lower ($P<0.01$) in model group. The activity of complex I, IV in the brain was significantly higher in therapy and prevention group while the activity of complex II and II+III had no obvious change in each group. In model group, mitochondrial membrane potential ($P<0.01$) was lower while mtNOS activity ($P<0.05$) and the production of ROS ($P<0.05$) was higher as compared with normal group. In therapy and prevention group, mitochondrial membrane potential increased ($P<0.01$) while mtNOS activity ($P<0.05$) and the production of ROS ($P<0.05$) decreased compared with normal group. **CONCLUSION** Catalpol can inhibit the brain damage induced by rotenone by recovering the respiratory complex activities and membrane potential level, reducing ROS production and inhibiting NOS activity.

Key words [catalpol](#) [rotenone](#) [mitochondria](#)

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