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论文

mitoK$_{ATP}$在槲皮素抑制心肌细胞肥大中作用

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摘要：

目的 探讨线粒体三磷酸腺苷（ATP）敏感性钾通道（mitoK$_{ATP}$）在槲皮素抑制乳大鼠心肌细胞肥大中的作用。方法 利用体外培养模型，以苯肾上腺素（PE）10 μmol/L诱导心肌细胞肥大，在mitoK$_{ATP}$通道阻断剂5-羟基癸酸（5-HD）存在情况下，槲皮素设25、50、100 μmol/L 3个剂量；用Lowry法检测心肌细胞蛋白质含量，消化分离法及计算机图像分析系统检测心肌细胞体积，逆转录-聚合酶链反应（RT-PCR）法检测心肌细胞心房钠尿肽（ANP）的mRNA 表达，Western印迹法测定K$_{ATP}$通道K$_{ir}$6.2亚基表达。结果 与对照组比较，模型组大鼠心肌细胞总蛋白质含量[（27.45±2.45）μg]增加，细胞体积[（2 278±156）μm^3]增大，ANP的mRNA 表达增多，K$_{ir}$6.2表达无 明显变化；与模型组比较，槲皮素50、100 μmol/L组心肌细胞蛋白含量[分别为（21.15±1.39）、（19.28±2.23）μg]减少、细胞体积[分别为（1 656±230）、（1 398±298）μm^3]减小，K$_{ir}$6.2表达增加，呈剂量依赖性。而在加入5-HD后，槲皮素对PE诱导心肌肥大的抑制作用消失。结论 槲皮素可通过开放mitoK$_{ATP}$通道抑制 PE诱导的乳大鼠心肌细胞肥大。

关键词： 线粒体钾通道(mitoK$_{ATP}$) 槲皮素 苯肾上腺素(PE) 心肌肥厚

Role of mitoK$_{ATP}$ in inhibitory effect of quercetin on myocardial hypertrophy in neonatal rats

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Abstract:

Objective To study the contribution of mitochohondrial adenosine triphosphate (ATP)-sensitive potassium channel (mitoK$_{ATP}$) in the antihypertrophic effect of quercetin(Que) in neonatal rat ventricular myocytes.Methods Myocardial cells of neonatal rats were cultured *in vitro*.The hypertrophic myocytes were induced by phenylephrine(PE) 10 μmol·L^{-1} and then different doses of Que(25,50,100 μmol·L^{-1}) were administered.Antihypertrophic effects of Que stimulation under administration of 100 μmol/L of 5-hydroxydecanoic acid (5-HD),a specific blocker of mitochondial K$_{ATP}$ channel,was also observed.The total protein content was measured with Lowry method.The cardiomyocyte size was measured by computer photograph analysis system.The expression of atrial natriuretic peptide(ANP) mRNA was determined with reverse transcription polymerase chain reaction (RT-PCR).The expression of K$_{ir}$6.2 was determined by Western blot.Results PE increased the total protein content (27.45±2.45 μg),the cardiomyocytes size (2 278±156 μm^3),and the expression of ANP mRNA.Que (50,100 μmol/L)showed a dose-dependent function,with the reduction of total protein content (21.15±1.39 to 19.28±2.23 μg),cardiomyocytes size (1 656±230 to 1 398±298 μm^3),the expression of ANP mRNA,and the expression of K$_{ir}$6.2 in the absence of 5-HD.Conclusion The results suggest that the inhibitory effect of Que on PE-induced hypertrophy may involve mito K$_{ATP}$.

Keywords: mitoK$_{ATP}$ quercetin phenylephrine cardiac hypertrophy

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