

论著

槲皮素对多柔比星致乳大鼠心肌细胞抗氧化酶活性及Fas/FasL表达变化的影响

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摘要 目的 探讨槲皮素对多柔比星致乳大鼠心肌细胞损伤的保护作用及机制。方法 原代培养乳大鼠心肌细胞, 随机分为正常对照组、多柔比星 $1.72 \mu\text{mol} \cdot \text{L}^{-1}$ 损伤组和多柔比星+槲皮素25, 50及 $100 \mu\text{mol} \cdot \text{L}^{-1}$ 组。槲皮素与心肌细胞培养3 h后, 加入多柔比星继续培养24 h, 正常对照组仅加等量DMEM培养液。倒置显微镜下观察细胞生长状态, 比色法检测培养液中谷胱甘肽过氧化物酶(GSH-Px)和超氧化物歧化酶(SOD)的活性, 用RT-PCR和Western印迹法检测Fas和FasL的mRNA和蛋白的表达。结果 与正常对照组相比, 多柔比星损伤组心肌细胞生长状态差, GSH-Px和SOD的活性降低, Fas和FasL的mRNA和蛋白的表达均升高。槲皮素25, 50及 $100 \mu\text{mol} \cdot \text{L}^{-1}$ 均可拮抗多柔比星所致的上述变化: GSH-Px分别为 (76 ± 3) , (73 ± 4) , (71 ± 3) vs $(69 \pm 3) \text{kU} \cdot \text{L}^{-1}$; SOD活性分别为 (31 ± 2) , (29 ± 2) , (29 ± 2) vs $(26 \pm 2) \text{kU} \cdot \text{L}^{-1}$; Fas mRNA: 0.61 ± 0.11 , 1.04 ± 0.12 , 1.29 ± 0.11 vs 1.61 ± 0.16 ; FasL mRNA: 0.81 ± 0.07 , 1.24 ± 0.10 , 1.57 ± 0.09 vs 1.79 ± 0.11 ; Fas蛋白: 1.08 ± 0.12 , 1.54 ± 0.10 , 1.89 ± 0.11 vs 2.15 ± 0.15 ; FasL蛋白: 1.51 ± 0.08 , 1.70 ± 0.12 , 2.20 ± 0.09 vs 2.41 ± 0.26 。结论 槲皮素可减轻多柔比星致乳大鼠心肌细胞凋亡, Fas和FasL蛋白表达的减少是其可能机制之一。

关键词 槲皮素 多柔比星 心肌细胞 基因表达 蛋白表达 Fas/FasL

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Effects of quercetin on antioxidant activity and Fas/FasL expression in neonate rat cardiomyocytes injured by doxorubicin

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Abstract

OBJECTIVE To study the protective effect and mechanism of quercetin(Que) on doxorubicin(Dox)-induced damage to cardiomyocytes in neonate rats in terms of Fas/FasL and oxidative stress. **METHODS** Cultured neonatal rat cardiomyocytes were randomly divided into 5 groups: normal control group, Dox $1.72 \mu\text{mol} \cdot \text{L}^{-1}$ group, Dox+Que 25, 50 and $100 \mu\text{mol} \cdot \text{L}^{-1}$ groups. The cells in Dox+Que groups were pretreated with Que for 3 h and then co-incubated with Dox for 24 h. To observe the cell growth condition by inverted microscope, the activity of glutathion peroxidase(GSH-Px) and superoxide dismutase(SOD) was detected by chromatometry; the mRNA and protein expression of Fas and FasL was detected by RT-PCR and Western blotting, respectively. **RESULTS** Compared with normal control group, the activity of GSH-Px and SOD in Dox group decreased; and the mRNA and protein expression of Fas and FasL was up-regulated. Que 25, 50 and $100 \mu\text{mol} \cdot \text{L}^{-1}$ could act as an antagonist of changes induced by Dox: GSH-Px, (76 ± 3) , (73 ± 4) , (71 ± 3) vs $(69 \pm 3) \text{kU} \cdot \text{L}^{-1}$; SOD, (31 ± 2) , (29 ± 2) , (29 ± 2) vs $(26 \pm 2) \text{kU} \cdot \text{L}^{-1}$; Fas mRNA: 0.61 ± 0.11 , 1.04 ± 0.12 , 1.29 ± 0.11 vs 1.61 ± 0.16 ; FasL mRNA: 0.81 ± 0.07 , 1.24 ± 0.10 , 1.57 ± 0.09 vs 1.79 ± 0.11 ; Fas protein: 1.08 ± 0.12 , 1.54 ± 0.10 , 1.89 ± 0.11 vs 2.15 ± 0.15 ; FasL protein: 1.51 ± 0.08 , 1.70 ± 0.12 , 2.20 ± 0.09 vs 2.41 ± 0.26 . **CONCLUSION** Que may significantly inhibit the apoptosis induced by Dox in the cultured myocardial cells. The potential mechanism is that Que can down-regulate Fas and FasL protein expression.

Key words quercetin doxorubicin cardiomyocyte gene expression protein expression Fas/FasL

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