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环维黄杨星D对阿霉素致小鼠心脏毒性的保到：

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Title: Protective effect of cyclovirobuxine D on mice suffered from doxorubicin-induced cardiotoxicity

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关键词: 环维黄杨星D; 阿霉素; 心肌细胞; 凋亡; 心肌纤维化

Keywords: cyclovirobuxine D; doxorubicin; myocardial cells; apoptosis; myocardial fibrosis

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摘要: 目的 研究环维黄杨星D（cyclovirobuxine D，Cvb-D）对阿霉素（doxorubicin，DOX）所致小鼠心脏毒性的影响。 方法 52只C57小鼠分为4组（*n*=12）：正常对照组（Control）、Cvb-D组、DOX组和Cvb-D+DOX组，每天灌胃给予Cvb-D（1 mg/kg）或生理盐水，连续给药4d，第5天单次腹腔注射DOX（15 mg/kg）或生理盐水。心肌组织切片进行Masson’s 染色，光镜下观察各组小鼠心肌组织纤维化改变；测定血清乳酸脱氢酶（LDH）、肌酸激酶（CK）水平及组织Caspase-3活性；采用末端标记TUNEL法检测各组心肌细胞凋亡情况；利用Western blot法检测Bcl-2和Bax蛋白表达水平及其比值。 结果 与正常对

照组相比，Cvb-D预处理可明显抑制DOX引起的体质量下降（*P*<0.05）以及心肌纤维化等组织形态学改变，显著抑制DOX引起的LDH、CK及Caspase-3活性升高（*P*<0.05），缓解DOX引起的心肌细胞凋亡，并显著提高DOX诱导的Bcl-2/Bax蛋白表达比值降低（*P*<0.05）。 结论 Cvb-D对DOX心脏毒性具有保护作用，并能够改善DOX引起的细胞凋亡。

Abstract: Objective To observe the effect of cyclovirobuxine D (Cvb-D) on mice suffered from doxorubicin (DOX)-induced cardiotoxicity. Methods Adult C57 mice were randomly divided into four groups (*n*=12) including a control group, a Cvb-D group, a DOX group and a Cvb-D+DOX group. Mice were given Cvb-D (1 mg/kg) intragastrically once a day for 4 consecutive days, followed by a single intraperitoneal injection of DOX (15 mg/kg) or saline, and the body weight was recorded thereafter. Four days later, the serum levels of creatine kinase (CK) and lactate dehydrogenase (LDH) and the activity of myocardial caspase-3 were determined, and cardiac tissue sections were stained with Masson’s trichrome to observe the morphology of myocardium. Apoptotic cells in cardiac tissues were observed by TUNEL assay. The protein expression levels of Bcl-2 and Bax were determined by Western blotting, and the ratio of Bcl-2/Bax was calculated. Results Cvb-D administered alone had no obvious effect on mice as compared with the control group. Pretreatment with Cvb-D significantly rescued DOX-induced decrease of body weight in mice (*P*<0.05), and alleviated myocardial fibrosis. Cvb-D significantly inhibited DOX-induced increase in serum levels of LDH and CK and caspase-3 activity (*P*<0.05) in cardiac tissues, and relieved DOX-induced myocardial apoptosis. Cvb-D also improved the ratio of Bcl-2/Bax expression decreased by DOX. Conclusion Cvb-D has a protective effect against DOX-induced cardiotoxicity possibly by inhibiting cell apoptosis.

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