[an error occurred while processing this directive]

山东大学学报(医学版) 2013, 51(9) 26-30 DOI: ISSN: 1671-7554 CN: 37-1390/R

基础医学

黄芩苷对ApoE-/-小鼠动脉粥样硬化及VE-钙黏蛋白表达水平的影响

尹海鹏，刘向群，于昕，王敏

山东大学齐鲁医院干部保健科， 济南 250012

摘要：

目的 探讨在动脉粥样硬化模型(AS)发生发展过程中VE-钙黏蛋白表达变化的意义及黄芩苷抗AS作用的可能机制。方法 建立小鼠ApoE-/- AS模型，20只雄性ApoE-/-小鼠随机分为AS模型组及黄芩苷治疗组，每组10只；雄性C57BL/6小鼠10只为正常对照组。建模成功后，黄芩苷治疗组小鼠给予黄芩苷50mg/(kg·d)灌胃，AS模型组及正常对照组给予生理盐水灌胃，连续灌胃4周。12周后处死动物，检测血清炎症指标，以及主动脉核因子-κB(NF-κB)、VE-钙黏蛋白的表达水平。结果 模型成功后，小鼠主动脉NF-κB、VE-钙黏蛋白阳性表达及炎症因子水平与正常对照组比较明显升高(P＜0.01或P＜0.05)，黄芩苷处理后，治疗组的小鼠主动脉NF-κB、VE-钙黏蛋白表达及炎症因子水平显著降低(P＜0.01或P＜0.05)，VE-钙黏蛋白表达与NF-κB阳性表达及炎症因子水平呈正相关(r=0.77)。结论 黄芩苷对AS有保护作用，其机制可能是通过降低NF-κB的转录活性、下调炎症因子以及VE-钙黏蛋白的表达，从而减轻内皮细胞损伤实现的。

关键词： 动脉粥样硬化；炎症；黄芩苷； VE-钙黏蛋白；核因子-κB；

Effect of Baicalin on atherosclerosis ApoE-/- mice and VE-cadherin level

YIN Hai-peng， LIU Xiang-qun，YU Xin，WANG Min

Depatment of Cadre Care, Qilu Hospital of Shandong University, Jinan 250012, China

Abstract:

Objective To study the effect of VE-cadherin expressions in the development of atherosclerosis (AS) and the possible mechanism of Baicalin in anti-AS. Methods ApoE-/- mice model of atherosclerosis (AS) was established. 20 male ApoE-/- mice were randomly divided into the model group and the baicalin treatment group. Ten C57BL/6 mice were chosen in the normal control group. 50mg/(kg·d) baicalin was given to mice in treatment group, and physiological saline was given to the mice in AS model group and normal control group for 4 weeks. After 12 weeks， the mice were put to death and the expression level of the serum inflammation biomarkers， aortic NF-κB and VE-cadherin were detected. Results NF-κB of aortic artery cells in mice, VE-cadherin positive expression and inflammatory factor levels were obviously increased compared with the normal group (P<0.01 or P<0.05). After the treatment of baicalin, aortic artery cells NF-κB in mice, VE-cadherin expression and inflammatory factors in the treatment group were significantly reduced (P<0.01 or P<0.05), which showed that VE-cadherin expression, the NF-κB expression and inflammatory factor levels were positively related. Conclusion Baicalin could have certain protective effect and the mechanism may be related to reduction the NF-κB transcription activity, lower inflammatory factors and VE-cadherin, thereby reducing the injury of endothelial cell.

Keywords: Atherosclerosis; Inflammation; Baicalin; VE-cadherin; Nuclear Factor kappa B

收稿日期 2013-06-01 修回日期 网络版发布日期

DOI:

基金项目:

山东省科技攻关计划项目(2007GGB01357)

通讯作者: 刘向群， Email：Liuxiangqun@medmail.com.cn

作者简介:

作者Email:

参考文献：

本刊中的类似文章