

痰瘀同治方含药血清对ox-LDL损伤人脐静脉内皮细胞 NF- κ B和ICAM-1表达的影响

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中文摘要:目的: 观察痰瘀同治方含药血清对氧化型低密度脂蛋白(ox-LDL)损伤的人脐静脉内皮细胞(HUVECs)核因子- κ B(NF- κ B)的活化和细胞间黏附分子-1(ICAM-1)表达的影响, 探讨痰瘀同治方抗动脉粥样硬化的分子机制。方法: SD大鼠随机分为正常组, 痰瘀同治方低、中、高剂量组(24, 48, 72 g·kg⁻¹·d⁻¹)和辛伐他汀组(18 mg·kg⁻¹·d⁻¹), 制备含药血清。体外培养HUVECs, 实验分为6组: ①正常组; ②模型组; ③痰瘀同治方低剂量组; ④痰瘀同治方中剂量组; ⑤痰瘀同治方高剂量组; ⑥辛伐他汀组。其中①、②组用20%正常鼠血清, ③~⑥组用20%各组含药血清, 除正常组外其余各组加入100 mg·L⁻¹ox-LDL刺激3 h或24 h后进行各项指标测定。Real-time PCR法检测HUVECs NF- κ B p65和ICAM-1 mRNA表达, Western blotting 检测ICAM-1蛋白表达, 细胞免疫荧光法检测NF- κ B p65核移位变化。结果: HUVECs经ox-LDL刺激后NF- κ B p65和ICAM-1的表达与正常组比较均明显升高($P < 0.01$)。痰瘀同治方和辛伐他汀含药血清能显著降低NF- κ B p65 mRNA表达及抑制其核移位($P < 0.05$), 降低ICAM-1 mRNA和蛋白表达($P < 0.05$), 其中以辛伐他汀和痰瘀同治方大剂量含药血清作用尤为显著($P < 0.01$)。结论: 痰瘀同治方能够通过抑制血管内皮细胞NF- κ B通路, 降低ICAM-1表达, 进而减少炎症反应, 这可能是其抗动脉粥样硬化分子机制之一。

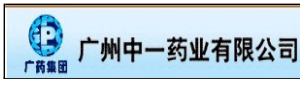
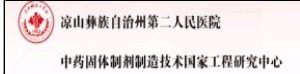
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Effects of Serum Containing Tanyu Tongzhi Fang on Expression of NF- κ B and ICAM-1 in HUVECs Injured by Ox-LDL

Abstract: Objective: To explore the anti-atherosclerosis (AS) effect of serum containing Tanyu Tongzhi Fang (TYTZF) on the activation of nuclear factor- κ B (NF- κ B) and the expression of intercellular adhesion molecule-1 (ICAM-1) in human umbilical vein endothelial cells (HUVECs) injured by oxidized low density lipoprotein (ox-LDL). Method: SD rats were equally divided into five groups in random: the control group, TYTZF low dose group, TYTZF middle dose group, TYTZF high dose group (24, 47, 72 g·kg⁻¹·d⁻¹) and simvastatin group (18 mg·kg⁻¹). After administration, the serum was taken for testing. HUVECs were treated with serum containing TYTZF and simvastatin respectively and incubated with ox-LDL (100 mg·L⁻¹) for an additional 3 h or 24 hours. The mRNA levels of NF- κ B p65 and ICAM-1 were measured by real-time PCR and the protein expression of ICAM-1 was detected by Western blotting. The activation of NF- κ B p65 was observed with immunofluorescence method. Result: The expression of NF- κ B p65 and ICAM-1 in serum containing TYTZF group and simvastatin group were significantly lower than that in model group ($P < 0.05$), especially in the high-dose serum containing TYTZF group and simvastatin group ($P < 0.01$). Conclusion: The mechanism of TYTZF anti-AS may be related to the inhibitory effect of NF- κ B pathway, thereby reducing the expression of ICAM-1 in vascular endothelial cells.

keywords: [Tanyu Tongzhi Fang](#) [atherosclerosis](#) [vascular endothelial cell](#) [NF- \$\kappa\$ B](#) [ICAM-1](#)

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