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痰瘀同治方含药血清对ox-LDL损伤人脐静脉内皮细胞 NF-κB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-κB(NF-κB)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^{-1}ox-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-κB p65和ICAM-1mRNA表达,Western blotting 检测ICAM-1蛋白表达,细胞免疫荧光法检测NF-κB p65核移位变化。 结果：HUVECs经ox-LDL刺激后NF-κB 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-κB 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-κB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。

中文关键词：痰瘀同治方　动脉粥样硬化　血管内皮细胞　核因子-κB　细胞间黏附分子-1

Effects of Serum Containing Tanyu Tongzhi Fang on Expression of NF-κB 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-kappa B(NF-κB) 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-κB 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-κB p65 was observed with immunofluorescence method. Result: The expression of NF-κB p65 and ICAM-1 in serum containing TYTZF group and simvastatin group were significantly lower than that in modle 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-κB pathway, thereby reducing the expression of ICAM-1 in vascular endothelial cells.

keywords: Tanyu Tongzhi Fang　atherosclerosis　vascular endothelial cell　NF-κB　ICAM-1

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