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论著

二苯乙烯苷对C反应蛋白诱导的小鼠巨噬细胞明胶酶A和B表达的影响

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摘要 目的 为探讨二苯乙烯苷(TSG)预防动脉粥样硬化斑块不稳定的可能机制，观察TSG对C反应蛋白（CRP）诱导的巨噬细胞表达明胶酶A和B的影响。方法 体外培养的小鼠腹腔巨噬细胞，分为空白对照组、模型组（给CRP 20 mg·L^{-1}）、模型+辛伐他汀100 μg·L^{-1}组、模型+TSG 120及60 μg·L^{-1}组。给予CRP 12 h后加入干预药物，共同培养24 h后进行指标的检测。用蛋白免疫印迹法观察各组细胞明胶酶A和B蛋白表达的差异；用逆转录聚合酶链反应法（RT-PCR）观察各组细胞明胶酶A和B mRNA表达的差异；ELISA法测定细胞培养液中白细胞介素6(IL-6)和肿瘤坏死因子α(TNFα)含量。结果 蛋白免疫印迹分析显示，模型组明胶酶A和B蛋白的表达较空白对照组明显增加；与模型组（明胶酶A：1.14±0.26，明胶酶B：1.26±0.24）相比，辛伐他汀（明胶酶A：0.71±0.12，明胶酶B：0.73±0.15）及TSG（120 μg·L^{-1}组：明胶酶A，0.74±0.11，明胶酶B，0.88±0.13；60 μg·L^{-1}组：明胶酶A，0.92±0.18，明胶酶B，1.12±0.18）均能降低明胶酶A和B蛋白的表达，并随TSG处理浓度的增加有下降趋势。RT-PCR显示，模型组明胶酶A和B的mRNA表达较空白对照组明显增加；与模型组（明胶酶A：2.45±0.18，明胶酶B：2.59±0.19）相比，辛伐他汀（明胶酶A：0.86±0.06，明胶酶B：0.98±0.10）及TSG（120 μg·L^{-1}组：明胶酶A，0.98±0.09，明胶酶B，1.24±0.13；60 μg·L^{-1}组：明胶酶A，1.32±0.12，明胶酶B，1.80±0.15）均能降低明胶酶A和B的mRNA表达，并随TSG处理浓度的增加有下降趋势。ELISA结果显示，与空白对照组比较，模型组IL-6和TNF-α水平明显升高；与模型组〔IL-6（614±52）ng·L^{-1}，TNFα(82.5±4.7)mg·L^{-1}〕相比，辛伐他汀〔IL-6(290±32)ng·L^{-1}，TNFα(36.3±2.7)mg·L^{-1}〕及TSG〔120 μg·L^{-1}组：IL-6 （310±28）ng·L^{-1}，TNFα(42.1±3.1) mg·L^{-1}；60 μg·L^{-1}组：IL-6（498±46）ng·L^{-1}，TNFα(58.6±3.4) mg·L^{-1}〕能明显降低细胞培养液中IL-6和TNFα含量。结论 TSG可通过抑制上调的明胶酶A和B的表达、IL-6及TNFα的分泌以抑制斑块不稳定。

关键词 二苯乙烯苷 C反应蛋白 明胶酶A 明胶酶B 白细胞介素6 肿瘤坏死因子

分类号 R967,R972.6

扩展功能

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Depressive effects of 2，3，5, 4′-tetrahydroxystilbene-2-*O*-β-*D*-glucoside on expressions of gelatinases A and B induced by C-reactive protein in mouse peritoneal macrophages

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Abstract

AIM To investigate the mechanism of 2, 3, 5, 4′-tetrahydroxystilbene-2-*O*-β-*D*-glucoside (TSG) on matrix remodeling in atherogenesis, the expressions of gelatinases A and B which were induced by C-reactive protein in macrophages were studied. **METHODS** Mouse peritoneal macrophages were cultured *in vitro* and intervened by different concentrations of TSG. There were 5 groups: normal control group, model group(recombinant human CRP, rhCRP 20 mg·L^{-1}), model+simvastatin 100 μg·L^{-1} group, model+TSG 120 and 60 μg·L^{-1} groups. After 24 h coincubation with drugs, gelatinases A and B proteins were determined by Western blot, and gelatinase A mRNA and gelatinase B mRNA were measured by reverse transcriptase polymerase chain reaction (RT-PCR). The level of IL-6 and TNFα were determined by ELISA. **RESULTS** Compared with the normal control, the expression of proteins of gelatinases A and B significantly increased in model group(gelatinase A: 1.14±0.26，and gelatinase B: 1.26±0.24). Compared with model group, simvastatin (gelatinase A, 0.71±0.12, and gelatinase B, 0.73±0.15) and TSG(120 μg·L^{-1} group: gelatinase A 0.74±0.11，and gelatinase B 0.88±0.13; 60 μg·L^{-1} group: gelatinase A 0.92±0.18，and gelatinase B 1.12±0.18) significantly decreased protein expressions. The inhibitory effect of TSG was increased with the concentration increased. Compared with normal control group, the expressions of mRNA of gelatinases A and B also significantly increased in model group (gelatinase A mRNA, 2.45±0.18，and gelatinase B mRNA, 2.59±0.19). The expressions of mRNA of gelatinases A and B were reduced markedly by simvastatin (gelatinase A: 0.86±0.06，and gelatinase B: 0.98±0.10) and TSG(120 μg·L^{-1} group: gelatinase A, 0.98±0.09, and gelatinase B: 1.24±0.13; 60 μg·L^{-1} group:gelatinase A, 1.32±0.12, and gelainase B, 1.80±0.15) in mouse peritoneal macrophages. The inhibitory effect of TSG was increased with the concentration increased. Compared with the normal

control, the level of IL-6 and TNFα remarkably increased in model group 〔IL-6 (614±52)ng·L^{-1}，and TNFα(82.5±4.7) mg·L^{-1}〕. The levels of IL-6 and TNF-α were decreased markedly by simvastatin〔IL-6 (290±32)ng·L^{-1}，and TNFα (36.3±2.7)mg·L^{-1}〕and TSG 〔120 μg·L^{-1} group: IL-6 （310±28）ng·L^{-1}，and TNFα (42.1±3.1)mg·L^{-1}; 60 μg·L^{-1} group: IL-6 （498±46）ng·L^{-1}，and TNFα (58.6±3.4)mg·L^{-1}〕 in mouse peritoneal macrophages. **CONCLUSION** TSG can prevent atherosclerosis plaques formation by down-regulating expressions of gelatinases A and B, and inhibit the levels of IL-6 and TNFα.

Key words tetrahydroxystilbene glucoside C-reactive protein gelatinase A gelatinase B interleukin-6 tumor necrosis factor

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