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论著

瑞巴匹特抑制脂多糖诱导肺上皮细胞株A549表达TLR4和释放TNF-α

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摘要
目的：研究瑞巴匹特对肺上皮细胞株A549 细胞TLR4表达及分泌肿瘤坏死因子（TNF-α）的影响。方法：用脂多糖(LPS)建立体外A549 细胞体外炎症损伤模型，实验分为对照组（无干预无刺激）、模型组（LPS刺激）、干预组1（LPS和10 mg/L瑞巴匹特）、干预组2（LPS和30 mg/L瑞巴匹特）、用ELISA观察A549细胞分泌TNF-α和RT-PCR及蛋白免疫印迹观察A549 细胞TLR4的表达。 结果：LPS诱导A549 细胞分泌TNF-α（与对照组比较，P＜0.01），在第6 h达高峰；LPS诱导A549 细胞表达TLR4（与对照组比较，P＜0.01）；瑞巴匹特可抑制LPS诱导A549 细胞分泌TNF-α和表达TLR4（与模型组比较，P＜0.05），但2组干预组间无统计学差异（P＞0.05）。 结论：瑞巴匹特的抗炎机制可能是通过抑制TLR4的表达，从而减少炎性细胞因子的释放；瑞巴匹特有可能作为一种有价值的抗感染治疗的辅助药物。

关键词 瑞巴匹特；TLR4；肿瘤坏死因子α 脂多糖 肺上皮细胞株
分类号

Rebamipide inhibited expression of TLR4 and TNF-α release in pulmonary epithelial cell line A549 induced by lipopolysaccharide

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Abstract
ObjectiveTo determine the effect of rebamipide on the expression of Toll-like recepter 4 (TLR4) and TNF-α release in pulmonary epithelial cell line A549.MethodsLipopolysaccharide (LPS) was used to induce A549 in vitro, which was divided into 4 groups: a control group, a model group(LPS), and 2 intervention groups (10 mg/L rebamipide plus LPS; 30 mg/L rebamipide plus LPS). TNF-α release was detected with ELISA and expression of TLR4 was detected with RT-PCR and Western blot.ResultsA549 cells were stimulated with LPS and TNF-α release was increased compared with the control group (P＜0.01), peaking at 6 h. Expression of TLR4 was also increased compared with the control group (P＜0.01)， but it was inhibited by rebamipide compared with the model group (P＜0.05). There was no significant difference between the 2 intervention groups (P＞0.05).ConclusionThe antiinflammatory mechanism of rebamipide may be reducing cytokine release by inhibiting TLR4 expression. Rebamipide may be used as a supplementary anti-infection drug.

Key words rebamipide Toll-like recepter 4 TNF-α lipopolysaccharide pulmonary epithelial cell line

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