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论著

沙眼衣原体激活MAPK通路而非NF-κB通路诱导IL-8生成

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摘要：

目的：研究沙眼衣原体诱导上皮细胞产生IL-8的信号通路。方法：应用Western印迹、免疫荧光技术和酶联免疫吸附实验（ELISA）检测沙眼衣原体感染Hela 229细胞后诱导的细胞内、外IL-8表达情况，并对细胞内IL-8定位；Western印迹和免疫荧光技术检测沙眼衣原体感染后MAPK 和NF-κB 信号通路活化情况；利用MAPK 和NF-κB 信号通路化学抑制剂抑制MAPK 和NF-κB 通路，观察其对沙眼衣原体诱导IL-8生成的影响。结果：沙眼衣原体感染可诱导IL-8产生，呈时间依赖性；沙眼衣原体感染可激活MAPK/ERK 和MAPK/p38 信号通路，但不能激活NF-κB信号通路；利用化学抑制剂抑制ERK和p38通路可以抑制沙眼衣原体诱导上皮细胞产生IL-8。结论：沙眼衣原体诱导上皮细胞产生IL-8依赖于MAPK 信号通路，但不依赖NF-κB信号通路。

关键词： 沙眼衣原体 MAPK通路 NF-&kappa B通路 IL-8

Induction of IL-8 by Chlamydia trachomatis through MAPK pathway rather than NF-κB pathway

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Abstract:

ObjectiveTo determine the signaling pathway required for Chlamydial induction of IL-8 expression in epithelial cells.MethodsThe production and localization of IL-8 in Chlamydia-infected Hela 229 cells were monitored using Western blot, immunoflourescence, and ELISA. Activation of MAPK and NF-κB signaling pathways were detected by Western blot and immunoflourescence. The effect of different signaling pathways on Chlamydia-induced Il-8 was measured by experiments of chemical inhibitors. ResultsIL-8 was induced by Chlamydia and was time-dependant. Chlamydial infection activated MAPK/ERK and MAPK/p38 pathways but not NF-κB pathway. Chlamydial induction of IL-8 was blocked by small molecule inhibitors targeting the ERK and p38 pathways. ConclusionChlamydia-induced IL-8 in cervical epithelial cells, the natural target cell type of Chlamydia trachomatis infection, is dependent on MAPK pathway but not NF-κB pathway, which provides important information for further understanding the molecular mechanism of Chlamydia-induced inflammatory pathologies.

Keywords: Chlamydia trachomatis；MAP kinase pathway；NF-κB pathway；IL-8

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