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曲古菌素A通过抑制MAPK/ERK通路上调食管癌EC1细胞CAR的表达 点此下载全文

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摘要：

摘 要 目的：观察曲古菌素A（trichostatin A，TSA）对人食管癌细胞EC1膜表面柯萨奇病毒-腺病毒受体(Coxsachievirus and adenovirus receptor, CAR)表达水平的影响，探讨MAPK/ERK信号通路在TSA上调CAR表达中的作用。方法：0.3、0.5、1.0 μmol/L的TSA处理EC1细胞48 h，采用免疫荧光、RT-PCR、Western blotting检测CAR的表达。以1.0 μmol/L TSA作用EC1细胞1、6、12、24、48 h，Western blotting检测p-ERK、CAR表达水平的变化，分析CAR表达和p-ERK水平变化的相关性。结果：0.3、0.5、1.0 μmol/L TSA处理EC1细胞后，CAR蛋白和mRNA水平均明显增加（P<0.05），并呈剂量依赖关系。1.0 μmol/L TSA作用EC1细胞6、12、24、48 h后，CAR蛋白表达较对照组均明显增加（P<0.05）；p-ERK表达水平均明显下降（P<0.05），两者变化呈显著负相关（r=-0.886，P<0.01）。结论：TSA能够上调人食管癌EC1细胞膜表面CAR的表达水平，其机制可能与其抑制MAPK/ERK通路有关。

关键词：食管癌细胞 曲古菌素A 柯萨奇病毒-腺病毒受体 细胞外信号调节激酶

Trichostatin A up-regulates expression of Coxsachievirus and adenovirus receptor in human esophageal cancer EC1 cells through inhibiting MAPK/ERK pathway Download Fulltext

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Abstract:

Abstract Objective:To observe the effect of trichostatin A (TSA) on Coxsachievirus and adenovirus receptor (CAR) expression in membrane of human esophageal cancer EC1 cells, and to discuss the role of MAPK/ERK signal pathway in the up-regulation of CAR expression triggered by TSA. Methods: EC1 cells were treated with TSA (0.3, 0.5, 1.0 μmol/L), and CAR expressions on EC1 cells were examined by immunofluorescence staining, RT-PCR and Western blotting analysis. EC1 cells were also treated with 1.0 μmol/L TSA for 0, 1, 6, 12, 24, and 48 h, and then the CAR expression and phosphorylation of ERK were detected by Western blotting analysis. The correlation between ERK phosphorylation level and the CAR expression was analyzed. Results: CAR protein and mRNA expressions in EC1 cells were significantly increased after treatment with 0.3, 0.5, and 1.0 μmol/L TSA (P<0.05), and the increase was in a dose-dependent manner. EC1 cells treated with 1.0 μmol/L TSA for different time periods also showed significantly increased CAR expression (P<0.05), while p-ERK expression levels in EC1 cells were remarkably decreased. The expression of p-ERK in EC1 cells treated with TSA was negatively correlated with that of CAR (r=-0.886, P<0.01). Conclusion: TSA can increase the expression of CAR in human EC1 cells, and the possible mechanisms may be related to the inhibition of ERK/MAPK pathway in EC1 cells.

Keywords:esophageal cancer cell trichostatin A Coxsachievirus and adenovirus receptor(CAR) extracellular signal-regulated kinase (ERK)

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