肿瘤防治研究 2007, Vol. 34 Issue (5): 359-362 DOI:
基础研究

p38MAPK 在结肠癌细胞凋亡中的作用及与COX-2 的关系

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Role of p38MAPK in Apoptosis of Colon Cancer Cells and Its Relationship with COX-2

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全文: PDF (148 KB) HTML (0 KB) 输出: BibTeX | EndNote (RIS) 背景资料

摘要 目的 探讨结肠癌细胞p38MAPK介导celecoxib（COX-2选择性抑制剂）抗肿瘤的作用及与COX-2的关系。方法 用MTT法检测celecoxib对人结肠癌HT-29细胞生长的作用，用Western blot法测定各组细胞COX-2和Phosph—p38MAPK蛋白表达量，采用流式细胞术检测celecoxib和SB203580（p38MAPK特异性抑制剂）作用后HT-29细胞凋亡和细胞周期分布。结果 p38MAPK和COX-2蛋白表达量与对照组（0．23±0.12）（0.95±0．14）相比，celecoxib可使p38MAPK蛋白表达水平明显升高（0．62±0．11），而使COX-2蛋白表达水平降低（0、44±0．11）；SB203580使p38MAPK（0.12±0．05）及COX-2蛋白（0、23±0．13）表达水平均降低；SB203580和celecoxib共同作用后，p38MAPK表达量介于celecoxib和SB203580作用之间（0．43±0．12），COX-2表达量下降最为显著（0．15±0．10））。celecoxib和eeleeoxib＋SB203580均可显著诱导HT-29细胞凋亡（P〈0．01和P〈0．05），与对照组（4．31%）相比，其凋亡率分别为40．95%、26．24%。结论 在HT29细胞中，celecoxib可通过活化p38MAPK而诱导结肠癌细胞凋亡，p38MAPK是COX-2的上游激酶，COX-2的表达水平受p38MAPK调控，并且COX-2可能对p38MAPK有负反馈调节作用。celecoxib是通过COX-2及其以外的p38MAPK通路诱导肿瘤细胞凋亡而发挥抗肿瘤作用的。

关键词： 结肠癌细胞株 COX-2 p38MAPK 信号转导 凋亡

Abstract： Objective To investigate the role of p38MAPKin mediating celecoxib (COX-2 selective inhibitor) inhibited the growth of tumor in colon cancer cells and it s relationship COX-2. Methods The cell gowth activity of HT-29 cells after the treatment by celecoxib was observed by MTT assay , flow cytometry was used to observed the effect of celecoxib and SB203580 (p38MAPK specific inhibitor) on apoptosis and the cell cycle dist ribution of HT-29 cells , the expression of Phosph-p38MAPK and COX-2 protein was detected by Western blot . Results Compared with the expression of p38MAPK(0. 23 ±0. 12) and COX-2 (0. 95 ±0. 14) of control group , p38MAPK expression (0. 62 ±0. 11) was higher than control group , while the expression of COX-2 (0. 44 ±0. 11) was lower than cont rol group which was treated by celecoxib. SB203580 could decrease the expression of p38MAPK(0. 12 ±0. 05) and COX22 (0. 23 ±0. 13) ;the expression of p38MAPK(0. 43 ±0. 12) was lower than control group , which was between celecoxib and SB203580 , the decrease of COX-2 was most significant (0. 15 ± 0. 10) . Compared with the apoptosis of cont rol group (4. 31 %) , celecoxib and celecoxib + SB203580 induced apoptosis significantly (P < 0. 01 and P < 0. 05) , the apoptosis rate of them was 40. 95 %、26. 24 % respectively. Conclusion Celecoxib can induced the apoptosis of human colon cancer HT-29 cell lines , which may through the activation of p38MAPK. In signal t ransduction of HT-29 cell lines , the upst ream kinase of COX-2 is p38MAPK, the expression of COX-2 was regulation by p38MAPK, which has the effect of degenerative feedback regulation by COX-2. Celecoxib induced the apoptosis of tumor cells and play the role of anti-neoplasiasm through COX-2 and p38MAPK.

Key words： Colon cancer cell lines COX-2 p38MAPK Signal transduction Apoptosis

收稿日期: 2006-03-31;

通讯作者: 韩彩丽

引用本文:

宋伟庆,刘　玉,韦金英等. p38MAPK 在结肠癌细胞凋亡中的作用及与COX-2 的关系[J]. 肿瘤防治研究, 2007, 34(5): 359-362.

SONG Wei-qing,LIU Yu,WEI Jin-ying et al. Role of p38MAPK in Apoptosis of Colon Cancer Cells and Its Relationship with COX-2[J]. CHINA RESEARCH ON PREVENTION AND TREATMENT, 2007, 34(5): 359-362.

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