[1]叶钧,宋丽丽,刘韵,等.肠分化细胞黏蛋白O-型糖链合成抑制导致MUC2表达减低以及对细菌侵袭易感[J].第三军医大学学报,2013,35(19):2056-2059.

Ye Jun,Song Lili,Liu Yun,et al.Inhibition of O-glycan chains synthesis in intestinal differentiated epithelial cells induces lower MUC2 expression and susceptibility to bacterial invasion[J].J Third Mil Med Univ,2013,35(19):2056-2059.

肠分化细胞黏蛋白O-型糖链合成抑制导致MUC2表达减低以及对细菌侵袭易感(PDF)

《第三军医大学学报》[ISSN:1000-5404/CN:51-1095/R] 卷: 35 期数: 2013年第19期 页码: 2056-2059 栏目: 论著 出版日期: 2013-10-15

Title: Inhibition of O-glycan chains synthesis in intestinal differentiated epithelial cells induces lower MUC2 expression and susceptibility to bacterial invasion

作者: 叶钧; 宋丽丽; 刘韵; 田音; 潘琼; 彭志红; 汪荣泉
第三军医大学西南医院全军消化病研究所

Author(s): Ye Jun; Song Lili; Liu Yun; Tian Yin; Pan Qiong; Peng Zhihong; Wang Rongquan
Institute of Gastroenterology, Southwest Hospital, Third Military Medical University, Chongqing, 400038, China

关键词: O-型糖链; benzyl-α-GalNAc; HT-29-Gal细胞

Keywords: O-glycan chains; benzyl-α-GalNAc; HT-29-Gal ; cells

分类号: R322.45; R329.26; R392.32

文献标志码: A

摘要: 目的 探讨O-型糖链合成的抑制对肠分化细胞内细菌侵袭数量和细胞内MUC2表达水平的影响。 方法 对肠分化细胞（HT-29-Gal）用O-型糖链抑制剂（benzyl-N-acetyl-α-D-galactosaminide，benzyl-α-GalNAc）处理，采用Real-time PCR和Western blot检测MUC2 mRNA和蛋白的表达情况。并将HT-29-Gal细胞及benzyl-α-GalNAc 处理的HT-29-Gal细胞分别与肠致病性大肠杆菌（enteropathogenic *E.coli*，EPEC）和肠出血性大肠杆菌（EHEC O157：H7） 37 ℃孵育2 h， 再加入100 μg/mL的庆大霉素，杀灭细胞外及粘附于细胞表面的细菌。最后采用系列稀释克隆计数法观察benzyl-α-GalNAc 处理的HT-29-Gal细胞对细菌侵袭的影响。 结果 Real-time PCR和Western blot检测发现经benzyl-α-GalNAc处理的HT-29-Gal细胞MUC2的mRNA和蛋白表达水平明显降低（$P<0.05$）。侵袭入benzyl-α-GalNAc 处理的HT-29-Gal细胞的EPEC和EHEC O157：H7的数量较对照细胞显著增加（$P<0.05$)。 结论 抑制HT-29-Gal细胞黏蛋白O-型糖链的合成导致侵袭入细胞内细菌数量增加和MUC2的表达降低。

Abstract: Objective To determine the effect of inhibiting O-glycan chains synthesis in intestinal differentiated epithelial cells on the bacteria invasion into the cells and MUC2 expression. Methods The intestinal differentiated cell line HT-29-Gal was treated with O-glycan chains synthesis inhibitor, benzyl-N-acetyl-α-D-

galactosaminide (benzyl-α-GalNAc). The expression of MUC2 at mRNA and protein levels was detected by real-time PCR and Western blotting. The HT-29-Gal cells treated with or without benzyl-α-GalNAc were co-cultured with *enteropathogenic E.coli* (EPEC) and *enterohemorrhage E.coli* (EHEC O157：H7) for 2 h, then the gentamicin (100 μg/mL) was added in the culture medium for 2 h to exclude extracellular and cellular surface-adherent bacteria after 3 times of washing with PBS. Finally, serial dilution cloning counting was used to observe the numbers of invaded bacteria into the HT-29-Gal cells treated with or without benzyl-α-GalNAc. Results Real-time PCR and Western blotting indicated that the mRNA and protein Levels of MUC2 were significantly lower in HT-29-Gal cells treated with the benzyl-α-GalNAc than the untreated HT-29-Gal cells (P<0.05).The numbers of EPEC and EHEC O157：H7 invaded into the HT-29-Gal cells exposed to benzyl-α-GalNAc were significantly larger than those untreated (P<0.05). Conclusion Inhibition of O-glycan chains synthesis in HT-29-Gal cells leads more bacteria to invade into the cells and lower expression of MUC2 at both mRNA and protein levels.

参考文献/REFERENCES:

叶钧,宋丽丽,刘韵,等. 肠分化细胞黏蛋白O-型糖链合成抑制导致MUC2表达减低以及对细菌侵袭易感[J].第三军医大学学报,2013,35(19):2056-2059.

更新日期/Last Update: 2013-09-30