中国病理生理杂志 2008 24 (2): 279-284 ISSN: 1000-4718 CN: 44-1187/R

论著

腺病毒介导的mPPARγ1基因转染抑制IFN-γ诱导ECV304细胞galectin-9基因和蛋白表达

胡琴[1],张宪军[2],蒋桂花[1],张运[1]

山东大学齐鲁医院 1心内科，国家教育部和卫生部心血管重构和功能研究重点实验室,2皮肤科,山东 济南 250012

收稿日期 2006-8-16 修回日期 2006-10-13 网络版发布日期 2008-11-16 接受日期 2006-10-13

摘要 目的：观察腺病毒介导的mPPARγ1转染抑制IFN-γ诱导ECV304细胞galectin-9基因和蛋白表达。方法：构建表达小鼠PPARγ1基因的复制缺陷型腺病毒表达载体；将融合80%的ECV304细胞给予不同刺激量（1×104 U/L、5×104 U/L、1×105 U/L和2×105 U/L）的IFN-γ干预；将IFN-γ（1×105 U/L）预刺激并孵育 24 h 的ECV304细胞分成对照组（C）、PPARγ基因过度表达组(P)、PPARγ活化剂曲格列酮干预组(T)以及PPARγ基因过度表达和曲格列酮共刺激组(PT)进行干预，观察不同剂量IFN-γ对ECV304细胞galectin-9基因和蛋白表达的作用，以及PPARγ基因过度表达和/或活化对上述作用的影响。结果：正常ECV304细胞galectin-9基因表达弱。IFNγ孵育24 h后， ECV304细胞galectin-9基因和蛋白表达增加，且galectin-9表达与IFN-γ具有量效关系。PPARγ1基因转染抑制IFN-γ诱导galectin-9基因/蛋白表达，曲格列酮对上述作用无影响；PPARγ1基因转染和曲格列酮共刺激抑制IFN-γ诱导galectin-9基因/蛋白表达与单一PPARγ1基因转染效应相似。正常ECV304细胞PPARγ表达量低，而PPARγ基因过表达和活化不影响内源性PPARγ基因表达。结论：PPARγ1基因转染抑制IFN-γ诱导ECV304细胞galectin-9基因/蛋白表达可能是PPARγ基因发挥免疫调控作用的一个重要机制。

关键词 过氧化物酶体增剂活化受体γ； 动脉硬化； Galectin-9

分类号 R363

Adenovirus-mediated mPPARγ1 gene overexpression inhibits IFN-γ-induced galectin-9 gene and protein expression in ECV304

HU Qin[1],ZHANG Xian-jun[2],JIANG Gui-hua[1],ZHANG Yun[1]

1Department of Cardiology, Key Laboratory of Cardiovascular Remodeling and Function Research, Chinese Ministry of Education and Chinese Ministry of Health, 2 Department of Dermatology, Qilu Hospital, Shandong University, Jinan 250012, China. E-mail: annettehu@163.com

Abstract

<FONT face=Verdana>AIM:To investigate whether adenovirus-mediated mPPARγ1 gene overexpression inhibits IFN-γ-induced galectin-9 gene and protein expression in ECV304. METHODS:A replication-deficient recombinant adenovirus expression vector of mPPARγ1 was constructed by using the AdEasy system. ECV304 were incubated for 24 h with 1×104 U/L, 5×104 U/L, 1×105 U/L and 2×105 U/L IFN-γ, respectively. ECV304 stimulated with 1×105 U/L IFN-γ were divided into 4 groups in random: P group (PPARγ1 gene overexpression), T group (treated with troglitazone 40 μmol/L in DMSO), PT group (PPARγ1 gene overexpression+troglitazone treatment) and control group. Changes of PPARγ and galectin-9 in mRNA and protein levels in different groups and subgroups were investigated by RT-PCR and immunoblotting. RESULTS: Galectin-9 expression was very few in normal ECV304. IFN-γ induced the expression of galectin-9 in ECV304. Degree of galectin-9 expression increased with the dose of IFN-γ. PPARγ1 gene overexpression inhibited IFN-γ-induced galectin-9 expression in ECV304. Galectin-9 mRNA and protein expressions from PT group and P group were inhibited in similar degree (P>0.05). However, this effect was not observed in troglitazone intervention (P>0.05). PPARγ expression was also very few in normal ECV304. PPARγ1 gene overexpression/activation had no effect on endogenous mPPARγ expression. CONCLUSION: This may partly contributed to the anti-inflammatory and immuno-

regulatory effect of PPARγ1 gene overexpression by inhibiting IFN-γ-induced galectin-9 gene and protein expression in ECV304.</FONT>

Key words Peroxisome proliferator-activated receptor γ Arteriosclerosis Galectin-9

DOI: 1007-4718

通讯作者 胡琴 annettehu@163.com