中国病理生理杂志 2007 23 (12): 2410-2413 ISSN: 1000-4718 CN: 44-1187/R

论著

蛋白激酶R与丙型肝炎病毒核心蛋白相互作用区域定位

颜学兵,季芳,傅涓涓,汪莉萍,梅蕾,潘修成,韩方正,张言超

徐州医学院附属第一医院感染病科，江苏 徐州 221002

收稿日期 2006-5-8 修回日期 2006-8-8 网络版发布日期 2008-8-29 接受日期 2006-8-8

摘要 目的： 观察丙型肝炎病毒（HCV）核心蛋白（CP）对蛋白激酶R(PKR)表达的影响；定位PKR与CP直接结合的区域。

方法: 对Huh-7、转染表达CP的Huh-7及含有全长HCV复制子(replicon) Huh-7细胞株的PKR表达水平及干扰素（IFN）诱导前后replicon Huh-7细胞中HCV结构蛋白和非结构蛋白表达水平作比较；对CP与PKR进行免疫共沉淀试验、谷胱苷肽S转移酶（GST）结合试验。

结果: Replicon Huh-7中PKR表达水平高于Huh-7及转染表达CP的Huh-7；IFN诱导后PKR表达增加，且明显抑制HCV结构和非结构蛋白的表达；PKR能与CP直接结合，依赖于PKR的N端1-180氨基酸（aa）。

结论: CP能直接作用于PKR N端1-180 aa，导致PKR组成性激活，从而干扰PKR介导的相关信号转导通路。CP与PKR的相互作用是HCV病毒蛋白与细胞蛋白相互作用又一新的模式，在HCV持续感染及肝癌2者发病机制方面可能起重要作用。

关键词 肝炎病毒,丙型 蛋白激酶R 病毒核心蛋白质类

分类号 R363

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Identification of domain of protein kinase R interacting with hepatitis C virus core protein

YAN Xue-bing,JI Fang,FU Juan-juan,WANG Li-ping,MEI Lei,PAN Xiu-cheng,HAN Fang-zheng,ZHANG Yan-chao

Department of Infectious Diseases,The First Affiliated Hospital,Xuzhou Medical College,Xuzhou 221002,China.E-mail:yxbxuzhou@126.com

Abstract

<FONT face=Verdana>AIM: To observe if hepatitis C virus (HCV) core protein (CP) influences the expression level of protein kinase R (PKR) and to map the direct interaction domain between PKR and CP.
METHODS: The expression levels of PKR in Huh-7,Huh-7 transfected with CP plasmid and replicon Huh-7 harboring selecting full length of HCV genome were studied.HCV structure and non-structure proteins in replicon Huh-7 with interferon (IFN) stimulation were compared.Co-immunoprecipitation and glutathione S-transferase (GST) binding assay were done between PKR and CP.
RESULTS: PKR expression level in replicon Huh-7 was higher than that in Huh-7 and Huh-7 transfected with CP expression plasmid.PKR was increased but structure and non-structure proteins in replicon Huh-7 were decreased after treated with IFN.The N-terminal 1-180 amino acid of PKR was the key binding site to CP.
CONCLUSION: CP directly binds to N-terminal 1-180 amino acid of PKR and leads to constitutive expression of PKR,which interferes signal transfer mediated by PKR.The interaction between CP and PKR might be a novel model of virus protein-cell protein interaction,which might play an important role in the pathogenesis of HCV persistent infection and hepatocellular carcinoma.</FONT>

Key words Hepatitis C virus Protein kinase R Viral core proteins

DOI: 1000-4718

通讯作者 颜学兵 yxbxuzhou@126.com