

首页 期刊概况 编委会 期刊内容 特

280~284.Polo样激酶3针对P73蛋白的磷酸化位点分析[J].桑梅香,刘丽华,丁春艳,孟 君,单保恩,中国肿瘤生物治疗杂志,20°

Polo样激酶3针对P73蛋白的磷酸化位点分析 点此下载全文

桑梅香 刘丽华 丁春艳 孟 君 单保恩

河北医科大学 第四医院 肿瘤研究所 免疫室,河北 石家庄 050011;河北医科大学 第四医院 科研中心,河北 石家庄 050 河北 石家庄 050011;河北医科大学 第四医院 科研中心,河北 石家庄 050011;河北医科大学 第四医院 肿瘤研究所 免涉学 第四医院 科研中心,河北 石家庄 050011

基金项目:河北省卫生厅科研基金资助项目(No. 20090155)

DOI: 10.3872/j.issn.1007-385X.2010.3.007

摘要:

目的:探讨P73蛋白上存在的能够被polo样激酶3(polo like kinases 3, Plk3)磷酸化的结构域或位点,并分析Plk3 免疫共沉淀法检测COS 7细胞中Plk3与P73蛋白之间的相互作用,荧光免疫染色法检测Plk3与P73蛋白在细胞中的定位。制外点突变法构建GST P73(1~130)点突变的P73(T86A)(第86位苏氨酸点突变为丙氨酸)质粒,体外磷酸化实验分析P73测PARP蛋白的裂解分析Plk3对P73介导人宫颈癌HeLa细胞凋亡的影响。结果:Plk3与P73蛋白之间存在相互作用,Plk3与获得不同的P73缺失突变体GST融合蛋白,Plk3在P73蛋白N端第63~113位氨基酸残基之间磷酸化P73蛋白。GST P73(1 氨酸(T86A)之后,不影响GST P73(1~130)蛋白的磷酸化状态。Plk3可抑制P73介导的HeLa细胞凋亡。结论:Plk3通过与13位氨基酸磷酸化,但第86位苏氨酸并非Plk3的特异作用位点;此外Plk3抑制P73介导的HeLa细胞凋亡。

关键词: polo样激酶3(Plk3) P73 磷酸化 位点 凋亡 宫颈癌细胞

Analysis of phosphorylated sites of P73 protein by polo like kinases 3 Download Fulltext

SANG Mei-xiang LIU Li-hua DING Chun-yan MENG Jun SHAN Bao-en

Department of Immunology, Tumor Research Institute, Fourth Hospital of Hebei Medical University, Shijia China; Research Center, Fourth Hospital of Hebei Medical University, Shijiazhuang, 050011, Hebei, China of Hebei Medical University, Shijiazhuang, 050011, Hebei, China; Research Center, Fourth Hospital of Hebei 050011, Hebei, China; Department of Immunology, Tumor Research Institute, Fourth Hospital of Hebei Medical University, Shijiazhuang, 05001

Fund Project: Project supported by the Scientific Research Foundation of Health Department of Hebei Programme Project: Project Supported by the Scientific Research Foundation of Health Department of Hebei Project: Project Supported by the Scientific Research Foundation of Health Department of Hebei Project: Project Supported by the Scientific Research Foundation of Health Department of Hebei Project: Project Supported by the Scientific Research Foundation of Health Department of Hebei Project: Project Supported by the Scientific Research Foundation of Health Department of Hebei Project: Project Supported by the Scientific Research Foundation of Health Department of Hebei Project Supported By P

Abstract:

Objective: To investigate the structural domains and sites of P73 which can be phosphorylated by pol analyze the effect of Plk3 on P73 mediated apoptosis. Methods: Co immunoprecipitation experiment was between Plk3 and P73. Immunofluorescence was used to examine the localization of Plk3 and P73 in cell P73 fusion protein were prepared. A site mutation plasmid of GST P73 (1 130) was constructed by conve (T86A) and was named GST P73 (1 130) (T86A). The phosphorylated domains and sites of P73 by Plk phosphorylation assay. The effect of Plk3 on P73 mediated apoptosis of HeLa cells was examined by cleacould interact with P73; Plk3 and P73 co located in the cell nuclei. Different deletion mutants of GST P73 prepared, and Plk3 phosphorylated P73 at N terminal 63 113 amino residues. Point mutation (T86A) of G not influence the phosphorylaten status of P73 by Plk3. Furthermore, Plk3 inhibited P73 mediated apopt can interact with and phosphorylate P73 at N terminal 63 113 amino residues (but not at the 86 threonin mediated apoptosis of HeLa cells.

Keywords: polo like kinases 3 (Plk3) P73 phosphorylation site apoptosis cervical cancer cell

查看全文 查看/发表评论 下载PDF阅读器