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48~51.miRNA-100下调polo样激酶1表达促进肝癌HepG2细胞凋亡[J].张红鸽,范秉琳,嵇晓辉,蔡新华,朱武凌.中国肿瘤生物miRNA-100下调polo样激酶1表达促进肝癌HepG2细胞凋亡 点此下载全文

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摘要:

目的: 探讨microRNA-100 (miR-100) 对人肝癌HepG2细胞中polo样激酶1 (polo-like kinase 1, Plk1) 表达 tamine介导,将miR-100 mimics转染入HepG2细胞中,RT-PCR和细胞免疫荧光法检测 Plk1 mRNA和蛋白的表达,并采 胞的凋亡情况。 结果: miR-100 mimics成功转染HepG2细胞,转染效率为(88.75±2.22)%。转染48 h后,miR-10 表达水平明显低于阴性对照组、空白对照组和脂质体组\[(0.71±0.01) vs (0.95±0.01)、(0.92±0.02)、(0.9: miR-100 mimics组Plk1蛋白几乎不表达,同时其细胞凋亡率明显高于阴性对照、空白对照组和脂质体组\[(26.95±6.72)3 71)%、(9.00±3.37)%,均P<0.05\]。 结论: miR-100能够抑制 Plk1 基因的表达,从而促进肝癌HepG2细胞

关键词: 肝癌 HepG2细胞 microRNA-100 Polo样激酶1 凋亡

miRNA-100 promotes hepatic carcinoma HepG2 cell apoptosis through down-regulating polo-like ki $\underline{\text{Fulltext}}$

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Abstract:

Objective: To explore the effects of microRNA-100 (miR-100) on expression of polo-like kinase 1 (Plk hepatic carcinoma HepG2 cells. Methods: HepG2 cells were transfected with miR-100 mimics by oligofects immunofluorescence were used to analyze Plk1 mRNA and protein expressions in HepG2 cells, respective HepG2 cells was detected by Annexin V-FITC kit. Results: HepG2 cells were successfully transfected by m efficiency was (88.75 \pm 2.22) %. 48 h after transfection, the expression of Plk1 mRNA decreased significations fransfection group compared with the negative control, blank control, and liposome groups (\[0.71 \pm 0.01 \\[0.93 \pm 0 02\], P<0.01). 72 h after transfection, Plk1 protein expression was almost undetectable in Hemimics. Meanwhile, the cell apoptosis rate in the miR-100 mimics group was significantly increased in concontrol, blank control, and liposome groups (\[0.26.95 \pm 6.72\]% vs \[15.03 \pm 5 12\]%, \[6.88 \pm 3.71\]%, \[Conclusion: miR-100 can inhibit the expression of Plk1 gene, therefore promoting the apoptosis of hepatic

Keywords: hepatic carcinoma HepG2 cell microRNA-100 polo-like kinase 1 apoptosis

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