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siRNA沉默整合素连接激酶对人膀胱癌细胞凋亡的影

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Title: Integrin-linked kinase siRNA induces apoptosis in human bladder cancer BIU-87 cells

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关键词: 整合素连接激酶; 膀胱癌; 凋亡

Keywords: integrin linked kinase; bladder cancer; apoptosis

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摘要: 目的 探讨应用整合素连接激酶（integrin-linked kinase, ILK）特异性siRNA沉默*ILK*基因的表达对人膀胱癌细胞凋亡的影响。 方法 将构建好的针对*ILK*基因的1个特异性siRNA表达质粒和1个无同源性的阴性对照质粒，在脂质体介导下稳定转染人膀胱癌BIU-87细胞，并将实验分为BIU-87 si ILK、BIU-87 vector及BIU-87细胞3组，运用TUNEL凋亡检测试剂盒和流式细胞术检测BIU-87细胞的凋亡，进一步运用Western blot检测siRNA ILK对凋亡相关蛋白Caspase-3、Bax和Bcl-2的影响。 结果 TUNEL检测结果显示，BIU-87 si ILK组出现大量凋亡细胞，而BIU-87细胞组和BIU-87 vector组仅有少量凋亡细胞出现。流式细胞仪检测结果显示，BIU-87 si ILK组细胞大约有（75.70±2.00）%的凋亡细胞，而BIU-87 vector组和BIU-87细胞组仅有（0.88±0.10）%和（1.66±0.90）%的凋亡细胞，Western blot结果显示，在BIU-87 si ILK组中，Bcl-2的表达明显降低（$P<0.05$），而Bax和Caspase-3的表达明显升高（$P<0.01$）。 结论 siRNA ILK可能通过调节凋亡相关蛋白Bcl-2、Bax及Caspase-3的表达诱导膀胱癌BIU-87细胞凋亡的发生。

Abstract: Objective To determine the effects of integrin-linked kinase (ILK) siRNA on the apoptosis of human bladder cancer BIU-87 cells. Methods Two siRNA vectors specific to ILK gene and one non-homologous negative control vector

were designed and constructed, then stably transfected into bladder cancer BIU-87 cells via Lipofectamine 2000, and were named as BIU-87 si ILK and BIU-87 vector. Cell apoptosis was assessed by TUNEL kit and flow cytometry. The expression of Caspase-3, Bax and Bcl-2 were assessed by Western blotting. Results The number of TUNEL-positive cells was significantly increased in the BIU-87 si ILK cells when compared with BIU-87 cells and BIU-87 vector cells. Flow cytometry showed that (75.70±2.00)% of counted cells became apoptotic in BIU-87 si ILK cells, however, about only (0.88±0.10)% and (1.66±0.90)% of counted cell became apoptotic in BIU-87 vector cells and BIU-87 cells groups respectively. The expression of Bcl-2 was significantly reduced in BIU-87 si ILK cells compared with BIU-87 vector and BIU-87 cells（P<0.05）, and that of Caspase-3 and Bax protein was stably increased (P<0.01）. Conclusion The siRNA ILK induces apoptosis in bladder cancer BIU-87 cells by regulating apoptosis-related proteins Caspase-3, Bax and Bcl-2.

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