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川芎嗪通过Akt信号通路影响前列腺癌PC3细胞的增殖和凋亡

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Title: Tetramethylpyrazine hydrochloride inhibits proliferation and apoptosis in human prostate cancer PC3 cells through Akt signaling pathway

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关键词: 川芎嗪; 前列腺肿瘤; 肿瘤细胞; 培养的; Akt

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摘要: 目的 研究盐酸川芎嗪对前列腺癌PC3细胞增殖和凋亡的影响及作用机制。 方 法 0、0.2、0.5、1.0、1.5、2.0、2.5、3.0、3.5 mg/mL盐酸川芎嗪处理雄激素非依 赖性前列腺癌PC3细胞, MTT法检测各组细胞的增殖能力, 荧光显微镜观察细胞核形态 学改变, Western blot检测Akt以及下游相关蛋白mTOR、p70S6蛋白及蛋白磷酸化的 表达, 以及凋亡相关蛋白Bcl-2和Bax的表达。 结果 盐酸川芎嗪能有效抑制前列 腺癌PC3细胞增殖, 且显示浓度时间依赖性($P<0.05$)。1.5、2.5 mg/mL的盐酸川芎嗪能 够降低Akt和p-Akt的表达, 进而下调 mTOR、p-mTOR、 p70S6及p-p70S6的表达 ($P<0.05$) ; 同时下调Bcl-2、上调Bax蛋白的表达($P<0.05$)。 结论 Akt及其 下游信号通路介导了盐酸川芎嗪抑制前列腺癌PC3细胞增殖及促凋亡作用。

Abstract: Objective To investigate the inhibitory effect of 2,3,5,6-tetramethylpyrazine hydrochloride (TMP) on the proliferation and apoptosis in human prostate cancer PC3 cells and to explore its underlying mechanism. Methods Different concentrations of TMP (0, 0.2, 0.5, 1.0, 1.5, 2.0, 2.5, 3.0 and 3.5 mg/mL) was added to treat PC3 cells. MTT assay was performed to test the inhibitory effect

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on cell proliferation, and fluorescence microscopy was applied to test the nuclear alteration. Western blot assay was used to detect the expression of Akt and the related downstream targets. Results TMP inhibited the proliferation in PC3 cells in a dose- and time-dependent manner ($P<0.05$) . TMP of 1.5 and 2.5 mg/mL decreased the protein levels of Akt, p-Akt, as well as the protein level of mTOR, p-mTOR, p70S6, and p-p70S6. Simultaneously, TMP also down-regulated the expression of Bcl-2, and up-regulated that of Bax ($P<0.05$) . Conclusion TMP inhibits the proliferation and induces the apoptosis in PC3 cells. Akt and the related downstream targets are involved in this process.

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