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Puma抑制卵巢癌细胞生长及其机制研究

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Title: Function and significance of Puma inhibiting ovarian carcinoma growth through inducing cell apoptosis

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关键词: Puma; 卵巢癌; 细胞凋亡

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摘要: 目的 探讨Puma对于卵巢癌细胞生长及体内成瘤的影响，了解其行使功能的分子机制。 方法 采用腺病毒载体实现Puma在卵巢癌细胞中的过表达，并通过CCK-8检测细胞增殖情况，通过Annexin-V染色测定细胞凋亡情况，通过小鼠荷瘤实验确定卵巢癌细胞体内成瘤能力。并进一步通过Western blot的方法检测了细胞凋亡相关基因Caspase-9和Bax蛋白的表达及定位情况。 结果 成功构建hTERT基因启动子介导的Puma基因腺病毒表达载体，并实现Puma基因在卵巢癌细胞中的过表达，过表达Puma后，卵巢癌细胞增殖被明显抑制（$P<0.05$），细胞凋亡增加，从对照组细胞的7.2%增加为过表达组的29%（$P<0.05$），且在小鼠体内的成瘤能力下降，所产生肿瘤组织的平均质量由0.53 g降至0.22 g（$P<0.05$）。 结论 Puma通过激活细胞内Caspase-9的活性、促使Bax从胞质转移至线粒体、促进细胞凋亡信号传导诱导细胞凋亡，从而抑制卵巢癌细胞增殖及体内成瘤能力。

Abstract: Objective To investigate the function and mechanism of p53 up-regulated modulator of apoptosis (Puma) in inhibiting ovarian carcinoma cell growth *in vitro* and *in vivo*. Methods Overexpression of Puma in ovarian carcinoma cell line COC1 was achieved by adenovirus vectors. Cell proliferation was detected using CCK-8 kit, meanwhile the percentage of apoptotic cells was identified by Annexin V staining. The mouse xenograft model was established to detect the inhibition on ovarian carcinoma cell growth *in vivo*. The expression of caspase 9 and Bax was detected by Western blotting. Results Puma was successfully

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over-expressed in COC1 cells using the established adenovirus expression vector. Over-expression of Puma inhibited the cell proliferation of COC1 cells evidently (P<0.05) and increased the percentage of apoptotic cells to 29% as compared to 7.2% in the control (P<0.05). Meanwhile, Puma inhibited the tumorigenicity of ovarian carcinoma cells *in vivo*. The average weight of xenografts was 0.22 g in the Puma overexpression group, significantly lower than 0.53 g in the control group (P<0.05). Conclusion Puma plays a tumor-suppressor role in ovarian carcinoma. Puma inhibits cell proliferation, induces cell apoptosis partially through activating caspase 9 and inducing Bax translocation to mitochondria and also inhibits the tumorigenicity *in vivo*.

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