[1]倪振洪,王滨,丁雯,等.左旋棉酚通过ERK通路诱导Daudi细胞发生自噬及意义[J].第三军医大学学报,2012,34(23):2384-2387.

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Title: (-)-gossypol induces autophagy in Daudi cells through ERK signal pathway

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关键词: 左旋棉酚; Burkitt淋巴瘤; 细胞增殖; 自噬; ERK

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摘要: 目的 探讨左旋棉酚诱导淋巴瘤Daudi细胞发生自噬可能机制及对细胞存活率的影响。 方法 采用CCK-8法检测左旋棉酚在体外对Daudi细胞增殖抑制作用的影响;采用台盼蓝排斥实验检测不同处理对细胞存活率的影响;Western blot检测细胞中自噬相关蛋白LC3、ERK和磷酸化ERK的表达情况;AO染色观察经左旋棉酚处理后Daudi细胞酸性小体的变化情况。 结果 左旋棉酚能剂量依赖性地抑制Daudi细胞的增殖和促进细胞死亡;AO染色后经左旋棉酚处理的细胞内可观察到大量的酸性小体形成;Western blot显示左旋棉酚能显著上调自噬相关蛋白LC3 Ⅱ的表达及增强磷酸化ERK的水平,抑制ERK的磷酸化能下调LC3 Ⅱ的表达;ERK抑制剂U0126及自噬抑制剂CQ和3-MA均能显著增强左旋棉酚的杀细胞效力。 结论 左旋棉酚可能通过ERK通路诱导Daudi细胞发生自噬,抑制ERK介导的自噬能够显著增强左旋棉酚的抗瘤效应。

Abstract: Objective To investigate the mechanism of (-)-gossypol-induced autophagy and its effect on cell viability in Burkitt lymphoma Daudi cells. Methods CCK-8 detection was used to assess the inhibitory effects of (-)-gossypol on the proliferation in Daudi cells. Trypan blue exclusion assay was used to detect cell viability during different treatments. Western blotting was used to determine the expression of LC3 Ⅱ and ERK. Acridine orange staining was used to detect the formation of acidic vesicular organelles (AVO). Results (-)-gossypol inhibited cell proliferation and induced cell death in a dose-dependent manner. Increased AVOs were noted after treatment of cells with (-)-gossypol. Western

blot analysis revealed that (-)-gossypol treatment markedly upregulated LC3 II and induced phosphorylation of ERK. Inhibition of ERK activity blocked the upregulation of LC3 II mediated by (-)-gossypol. Inhibition of autophagy by U0126 (ERK inhibitor), CQ and 3-MA (autophagy inhibitors) enhanced cell death mediated by (-)-gossypol. Conclusion (-)-gossypol effectively inhibits cell proliferation and induces autophagy in Burkitt lymphoma Daudi cells *via* ERK pathway.

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