

1, 6二磷酸果糖增强蛋白酶体抑制剂对人乳腺癌细胞MCF-7的抑瘤效应

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Combination of Fructose-1,6-Diphosphate and Proteasome Inhibitor Enhances Inhibition of Cell Proliferation in Human MCF-7 Breast Cancer Cells

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全文: PDF (683 KB) HTML (0 KB) 输出: BibTeX | EndNote (RIS) 背景资料

摘要 目的探讨抑制蛋白酶体活性是否可以诱导人乳腺癌细胞MCF-7的自我吞噬, 1,6二磷酸果糖对自我吞噬的影响及自我吞噬对细胞增殖的作用。方法采用MTT法检测细胞增殖, 蛋白质印迹检测自我吞噬相关蛋白LC3的表达。结果蛋白酶体抑制剂硼替佐米以剂量依赖方式抑制MCF-7细胞的增殖并诱导细胞的自我吞噬, 但是, 当1,6二磷酸果糖与硼替佐米联合应用后, 可逆转硼替佐米诱导的自我吞噬并增强硼替佐米对MCF-7细胞的增殖抑制。结论蛋白酶体活性的抑制可诱导人乳腺癌细胞MCF-7的自我吞噬代偿性激活, 1, 6二磷酸果糖可抑制激活的自我吞噬, 其与硼替佐米的联合应用可增强其抑瘤效应。

关键词: 蛋白酶体抑制剂 自我吞噬 细胞死亡

Abstract: Objective To investigate whether inhibition proteasome can induce autophagy, effects of fructose-1,6-diphosphate(FDP) on autophagy and effects of autophagy on the fate of human breast cancer MCF-7 cells. Methods Cell viability was measured by MTT assay. Apoptosis was detected by flow cytometry. The expression of autophagy related proteins was determined by Western blot. Results MCF-7 cells proliferation was inhibited by proteasome inhibitor Bortezomib in a dose dependent manner and autophagy was activated in the same manner. However, when MCF-7 cells were co-treated with Bortezomib and FDP, it could lead the most significant inhibition of cell proliferation. Moreover, FDP blocked the increase of LC3-II protein expression induced by Bortezomib. Conclusion The inhibition of proteasome can induce autophagy in human breast cancer MCF-7 cells and FDP could inhibit autophagy induced by proteasome inhibitor. Combination of FDP and Bortezomib increases cell death.

Key words: Proteasome inhibitors Autophagy Cell death

收稿日期: 2011-05-10;

引用本文:

姚峰,王冠楠,李娟娟等. 1, 6二磷酸果糖增强蛋白酶体抑制剂对人乳腺癌细胞MCF-7的抑瘤效应 [J]. 肿瘤防治研究, 2011, 38(12): 1360-1362.

YAO Feng,WANG Guan-nan,LI Juan-juan et al. Combination of Fructose-1,6-Diphosphate and Proteasome Inhibitor Enhances Inhibition of Cell Proliferation in Human MCF-7 Breast Cancer Cells[J]. CHINA RESEARCH ON PREVENTION AND TREATMENT, 2011, 38(12): 1360-1362.

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