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NVP-BEZ235增敏左旋棉酚杀伤肝癌HepG2细胞的作用及可能机制: 分享到:

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Title: Effect of NVP-BEZ235 on sensitization of HepG2 cells to (-)-gossypol

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摘要: 目的 探讨PI3K/mTOR抑制剂NVP-BEZ235增敏左旋棉酚[(-)-gossypol]杀伤肝癌细胞HepG2的作用及可能机制。 方法 用NVP-BEZ235、左旋棉酚或二者联合处理HepG2细胞, CCK-8法检测不同处理对细胞增殖的影响, 流式细胞术检测不同处理对细胞凋亡的影响, Western blot检测不同处理对细胞中mTOR磷酸化水平以及Mcl-1蛋白水平的影响。 结果 联合使用NVP-BEZ235和左旋棉酚可显著抑制HepG2细胞增殖, 并促进细胞凋亡, 其中左旋棉酚可上调HepG2细胞中mTOR磷酸化水平及Mcl-1蛋白水平, 导致抵抗发生, NVP-BEZ235能够剂量依赖性抑制mTOR磷酸化($P < 0.01$), 并抑制左旋棉酚对Mcl-1的上调作用。 结论 NVP-BEZ235可通过抑制mTOR进而下调Mcl-1来增强左旋棉酚杀伤HepG2细胞的效果。

Abstract: Objective To determine the effect and associated mechanisms of PI3K/mTOR inhibitor NVP-BEZ235 on the sensitization of HepG2

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cells to Bcl-2 inhibitor, (-)-gossypol. **Methods** HepG2 cells were treated with NVP-BEZ235, (-)-gossypol or combined these 2 agents. The anti-proliferation effects of different treatments were detected by CCK-8 assay and cell apoptosis was detected by flow cytometry. Protein level of myeloid cell leukemia-1 (Mcl-1) and phosphoralation levels of mTOR were detected by Western blotting. **Results** Compared to (-)-gossypol alone, the combination of NVP-BEZ235 and (-)-gossypol significantly inhibited the proliferation and induced the apoptosis in HepG2 cells. (-)-gossypol upregulated the protein expression of Mcl-1 and the phosphoralation of mTOR in HepG2 cells, while NVP-BEZ235 inhibited the phosphoralation levels of mTOR in dose-dependent manner and attenuated (-)-gossypol-induced Mcl-1 accumulation. **Conclusion** NVP-BEZ235 sensitized HepG2 cells to (-)-gossypol partly by inhibiting mTOR phosphoralation and