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新型低氧还原剂Q39通过阻断HIF-1 α 转运引起肝癌Bel-7402细胞凋亡

Novel Hypoxia-selective Compound Q39 Induced Bel-7402 Cell Apoptosis via Blocking HIF-1 α Translocation

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英文关键词: [Q39](#) [HIF-1 \$\alpha\$](#) [ERK1/2](#) [apoptosis](#) [anti-tumor activity](#)

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中文摘要:

目的 评价Q39低氧条件下诱导肝癌细胞Bel-7402细胞凋亡的抗肿瘤活性及其机制。方法 MTT法测定Q39对人肝癌细胞Bel-7402增殖抑制作用。PI染色法检测Q39诱导人肝癌细胞凋亡作用。免疫荧光法检测HIF-1 α 蛋白的转运和表达。结果 Q39在常氧和低氧下均抑制肝癌Bel-7402细胞的生长。Q39在低氧条件下促进Bel-7402肿瘤细胞凋亡。Q39通过抑制ERK1/2的磷酸化, 明显抑制HIF-1 α 的转运。结论 Q39在低氧条件下通过抑制ERK1/2信号通路引起Bel-7402肿瘤细胞凋亡。

英文摘要:

OBJECTIVE To study the anti-cancer activity and mechanism of Q39 in hypoxia. METHODS The anti-proliferation activity of Q39 were analyzed by MTT, apoptosis was detected by PI and flowcytometry analysis. HIF-1 α protein expression level and translocation was analyzed by immunostaining and WB. RESULTS The present study indicated that Q39 exerted anti-proliferative effects against human cancer cells Bel-7402 in hypoxia. Downregulation of ERK1/2 inhibited by Q39 resulted in blocking HIF-1 α translocation, which further induced the apoptosis of Bel-7402. CONCLUSION These findings build the rationale for further development of candidate compound Q39 against hepatoma(Bel-7402).

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