43~47.TRAIL联合紫杉醇对人脑胶质瘤U87细胞的抑制效应及其可能的机制[J].仇波,吴鹏飞,王勇,陶钧,欧绍武,王运杰.中国肿瘤生物治疗杂志,2013,20(1)

TRAIL联合紫杉醇对人脑胶质瘤U87细胞的抑制效应及其可能的机制 点此下载全文

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基金项目：国家自然科学基金资助（No. 81000565）

DOI：10.3872/j.issn.1007-385X.2013.1.007

摘要：

目的： 研究肿瘤坏死因子相关凋亡诱导配体（tumor necrosis factor-related apoptosis-inducing ligand，TRAIL）联合紫杉醇处理对人脑胶质瘤U87细胞的抑制效应及其可能的机制。 方法： MTT法检测紫杉醇组、TRAIL组和TRAIL/紫杉醇组对U87细胞增殖的抑制率；流式细胞术检测不同给药方案对U87细胞凋亡的影响；Western blotting检测不同处理后U87细胞TRAIL死亡受体（death receptor，DR）4、DR5以及caspase-8和caspase-3的表达水平。 结果： MTT结果显示，单独应用TRAIL或紫杉醇可有效抑制U87细胞的增殖，并呈浓度依赖性。TRAIL（500 ng/ml）/紫杉醇（0.5 μmol/L）联合给药组可协同抑制U87细胞的增殖，相互作用指数（coefficient of drug interaction，CDI）为0.59；且联合用药组对U87细胞增殖的抑制率明显高于TRAIL和紫杉醇单独用药组 \[（70.24±3.68）% vs （27.01±2.36）%，（21.31±4.85）%，P<0.01\]；TRAIL/紫杉醇联合用药组U87细胞凋亡率明显高于对照组、TRAIL组及紫杉醇组\[（67.67±2.46）% vs（1.80±1.13）%、（22.13±2.18）%、（35.90±2.53）%， P<0.01\]。TRAI与紫杉醇联合用药组U87细胞中DR4、caspase-8及caspase-3的表达比TRAIL组或紫杉醇组显著增加（ P<0.05），而DR5表达则无明显变化（P>0.05）。 结论： TRAIL联合紫杉醇处理通过上调DR4、caspase-8及caspase-3的表达，抑制U87细胞的增殖，诱导U87细胞凋亡

关键词：脑胶质瘤 U87细胞 肿瘤坏死因子相关凋亡配体 紫杉醇 凋亡

Inhibitory effects of paclitaxel combined with TRAIL on human glioma U87 cells and the possible mechanism Download Fulltext

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Fund Project:Project supported by the National Natural Science Foundation of China (No. 81000565)

Abstract:

Objective:To investigate the inhibitory effects of tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) combined with paclitaxel treatment on human glioma U87 cells and the possible mechanism. Methods: MTT assay was used to detect the proliferation inhibitory rates of U87 cells in the paclitaxel group, TRAIL group, and TRAIL/paclitaxel combination group, and flow cytometry was used to detect the effects of different treatments on apoptosis of U87 cells. The expression levels of TRAIL death receptor (DR) 4, DR5, caspase-8 and caspase-3 in U87 cells after different treatments were measured by Western blotting. Results: MTT results showed the TRAIL or paclitaxel used alone demonstrated a favorable inhibitory effect on proliferation of U87 cells in a concentration-dependent manner. Combined application of TRAIL (500 ng/ml) and paclitaxel (0.5 μmol/L) showed a synergistic inhibitory effect on the proliferation of U87 cells with the coefficient of drug interaction (CDI) being 0.59. The proliferation inhibitory rate of U87 cells in the combination group was significantly higher than that in the TRAIL or paclitaxel used alone groups (\[70.24±3 68\] % vs \[27 01±2.36\] %, \[21.31±4.85\] %, P<0.01). The apoptotic rate of U87 cells in the TRAIL/paclitaxel combination group was significantly higher than that in the control group, TRAIL group, or paclitaxel group (\[67.67±2.46\] % vs \[1.80±1.13\] %, \[22.13±2.18\] %, \[35.90±2.53\]%, P<0.01). The up-regulation expressions DR4, caspase-8 and caspase-3 in U87 cells was more obvious in TRAIL/paclitaxel combination treatment group than that in the TRAIL or paclitaxel groups (P<0.05). However, no obvious change in DR5 xpression was observed (P>0.05). Conclusion: TRAIL combined with paclitaxel treatment can up-regulate DR4, caspase-8 and caspase-3 expressions, thereby inhibiting the proliferation and inducing the apoptosis of U87 cells.

Keywords:glioma U87 cell TRAIL paclitaxel apoptosis

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