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人参皂苷Rb1拮抗达沙替尼抑制NK细胞杀伤卵巢癌

Ginsenoside Rb1 Antagonist Dasatinib-induced Inhibi
Cytotoxicity to Ovarian Cancer Cell

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中文关键词： 达沙替尼 人参皂苷Rb1 自然杀伤细胞 卵巢癌细胞 杀伤功能

英文关键词：dasatinib ginsenoside Rb1 natural killer cell ovarian cancer cell cytotoxicity

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中文摘要：

目的 研究抗癌药达沙替尼对自然杀伤细胞(NK细胞)杀伤卵巢癌细胞
苷Rb1拮抗这种免疫抑制效应的作用及其分子机制。方法 分别用达沙替尼、
联合人参皂苷Rb1预处理NK细胞，未经药物处理的NK细胞设为对照。采用CI
仪检测与NK细胞混合培养的卵巢癌HO-8910细胞的死亡率来明确NK细胞的杀
TC/PI双染色法测定NK细胞的凋亡率来明确其生存活力；采用real-time P
NA表达量；采用免疫印迹法检测NK细胞ERK的蛋白水平。结果 与对照组相
对卵巢癌HO-8910细胞的杀伤率显著下降(P<0.01)，NK细胞的颗粒酶B的mR
达均下调；达沙替尼合并人参皂苷Rb1处理组的NK细胞对卵巢癌HO-8910细
(P<0.05)，而且颗粒酶B的mRNA水平、pERK表达均较达沙替尼组有所恢复；
1单独处理组的NK细胞的生存活力、颗粒酶B水平、pERK表达量以及杀伤功
结论 达沙替尼对NK细胞杀伤卵巢癌细胞有抑制效应，可能与下调NK细胞的
磷酸化有关。人参皂苷Rb1虽然不能直接活化NK细胞，但是能拮抗达沙替尼
提示联用人参皂苷Rb1能减少抗癌药物达沙替尼对免疫效应细胞的抑制作用

英文摘要：

OBJECTIVE To analyze the inhibiting effects of dasatinib
cells cytotoxicity on ovarian cancer cell and explore the anta
ginsenoside Rb1 on dasatinib-induced immunosuppression effects
were pre-treated with dasatinib, ginsenoside Rb1 and dasatinib

ginsenoside Rb1, respectively. The NK cells with no drug pretr
as control. CFSE/PI double staining was used to measure the de
cancer cell HO-8910 cells after co-culturing with NK cells; An
was used to detect the vitality of NK cells; real-time PCR was
mRNA level of granzyme B and Western-blotting was used to dete
levels of ERK. RESULTS Compared with the non-pretreated NK cel
levels of granzyme B, phosphorylation levels of ERK and the cy
significantly down-regulated in dasatinib-treated NK cells (P<
dasatinib treated NK cells, pre-treated in the combination of
ginsenoside Rb1, the transcriptional levels of granzyme B, pER
cells were elevated(P<0.05). However, the levels of transcript
and cytotoxicity of NK cells between ginsenoside Rb1 treated g
showed no significant difference(P<0.05). CONCLUSION Dasatinib
on the cytotoxicity of NK cells by decreasing the amount of gr
crucial cytotoxic molecules in NK cells and inhibiting phospho
crucial for NK cells reactivity. Though ginsenoside Rb1 can't
directly, it has antagonistic effects on dasatinib-induced imm