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SOCS3 Reverses Multidrug Resistance by Inhibiting MDR1 in Mammary Cell Carcinoma

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Abstract : Suppressors of cytokine signalling (SOCS3), a newly indentified anti-apoptotic molecule is a downstream effecter of the receptor tyrosine kinase-Ras signalling pathway. Current study has uncovered that SOCS3 may have wide and imperative capacities, particularly because of its close correlation with malignant tumors. To investigate the impact of SOCS3 on MDR, we analyzed the expression of P-gp and SOCS3 by immune-histochemistry and found there was positive correlation between them. At that point we effectively interfered with RNA translation by the contamination of siRNA of SOCS3 into MCF7/ADM breast cancer cell lines through a lentivirus, and the expression of the target gene was significantly inhibited. After RNAi the drug resistance was reduced altogether and the expression of MDR1 mRNA and P-gp in MCF7/ADM cell lines demonstrated a significant decrease. Likewise the expression of P53 protein increased in a statistically significant manner ($p \leq 0.01$) after RNAi exposure. Moreover, flowcytometry analysis uncovers that cell cycle and anti-apoptotic enhancing capacity of cells changed after RNAi treatment. These outcomes proposed SOCS3 may take part in breast cancer MDR by managing MDR1 and P53 expression, changing cell cycle and enhancing the anti-apoptotic ability.

Keywords : SOCS3gene, breast cancer, multidrug resistance, MDR1 gene, RNA interference
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