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Erlotinib enhances the CIK cell-killing sensitivity of lung adenocarcinoma A549 cells

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ABSTRACT. We examined the effects and molecular mechanism of the epidermal growth factor receptor (EGFR) tyrosine kinase inhibitor erlotinib on NKG2D ligand expression in human lung adenocarcinoma A549 cells and the cytotoxicity of cytokine-induced killer cells. Flow cytometry was used to detect NKG2D ligand expression in A549 cells under effects of erlotinib and EGFR downstream molecules, including LY294002 (phosphoinositide 3-kinase inhibitor), SB203580 (mitogen-activated protein kinase inhibitor), and STAT21 (signal transduction and transcription 3 inhibitor) after 24 h. A lactate dehydrogenase release assay was used to detect, at different effector-to-target ratios, the A549 cell killing activity of cytokine-induced killer cells before and after treatment with 10 μM erlotinib. Erlotinib suppressed MICA expression in A549 cells and upregulated MICB and UL16 binding protein 1 expression. EGFR downstream molecules mitogen-activated protein kinase and signal transduction and transcription 3 inhibitor did not affect the expression of NKG2D ligands in A549 cells. The phosphoinositide 3-kinase inhibitor reduced MICA expression in A549 cells, while erlotinib enhanced the killing sensitivity of cytokine-induced killer cells in A549 cells. The anti-lung carcinoma effects of

EGFR tyrosine kinase inhibitor were associated with the sensitivity of lung cancer cells to enhanced immune cell killing.

Key words: Cytokine-induced killer cells; NKG2D ligands; EGFR tyrosine kinase inhibitor; Lung cancer

