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Pathogenetic pathways leading to glioblastoma multiforme: association between gene expressions and resistance to erlotinib.

[Löw S](#), [Vougioukas VI](#), [Hielscher T](#), [Schmidt U](#), [Unterberg A](#), [Halatsch ME](#).

Department of Neurosurgery, Ruprecht Karls University, Im Neuenheimer Feld 400, D-69120 Heidelberg, Germany.

BACKGROUND: The antiproliferative effects of erlotinib, an epidermal growth factor receptor (EGFR) tyrosine kinase inhibitor, on human glioblastoma multiforme (GBM) cell lines in vitro and in vivo are widely variable and independent of EGFR baseline expression levels, indicating that more complex genetic signatures may form the molecular basis of GBM response to erlotinib. This study sought to determine which genes within two common genetic pathways of GBM pathogenesis, i.e., the primary and secondary pathways, may be involved in mediating the cellular response of human GBM towards erlotinib. **MATERIALS AND METHODS:** Complementary (c)RNAs from cell lines selected to represent the sensitive, intermediately responsive and resistant phenotypes, respectively, were hybridized to CodeLink Human Whole Genome Bioarrays. **RESULTS:** Expression analysis of prospectively selected 104 genes pertaining to the primary and secondary pathways of GBM pathogenesis identified two genes (IGF1, PIK3C2B) the expression of which significantly correlated with cellular resistance towards erlotinib. **CONCLUSION:** Among the genes constituting two common pathways of GBM pathogenesis, two candidate genes may confer GBM resistance towards erlotinib, suggesting that resistance towards this compound may be acquired during the natural evolution of GBM.

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