The response of head and neck squamous cell carcinoma to cetuximab treatment depends on Aurora kinase A polymorphism.

The aim of this study was to evaluate the efficiency of cetuximab-based anti-EGFR treatment and Aurora kinase A / B knockdown as a function of Aurora kinase polymorphism in HNSCC cell lines. First, protein expression of Aurora kinase A / B and EGFR and Aurora kinase A polymorphism were studied in tumour samples. The survival and proliferation of Aurora kinase A homo- (Cal27) and heterozygous (HN) HNSCC cell lines was evaluated using a colony formation assay and a flow cytometric assay. Also, aneuploidy was determined. EGFR signalling pathway were visualised by western blotting. Immunohistochemistry revealed the overexpression of Aurora kinase A / B in HNSCC. The knockdown of each kinase caused a significant decrease in clonogenic survival, independent of Aurora kinase A polymorphism. In contrast, cetuximab treatment impaired clonogenic survival only in the Aurora kinase A-homozygous cell line (Cal27). This study provides in vitro evidence for the predictive value of Aurora kinase A polymorphism in the efficiency of cetuximab treatment. Resistance to cetuximab treatment can be overcome by simultaneous Aurora kinase A/B knockdown.
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