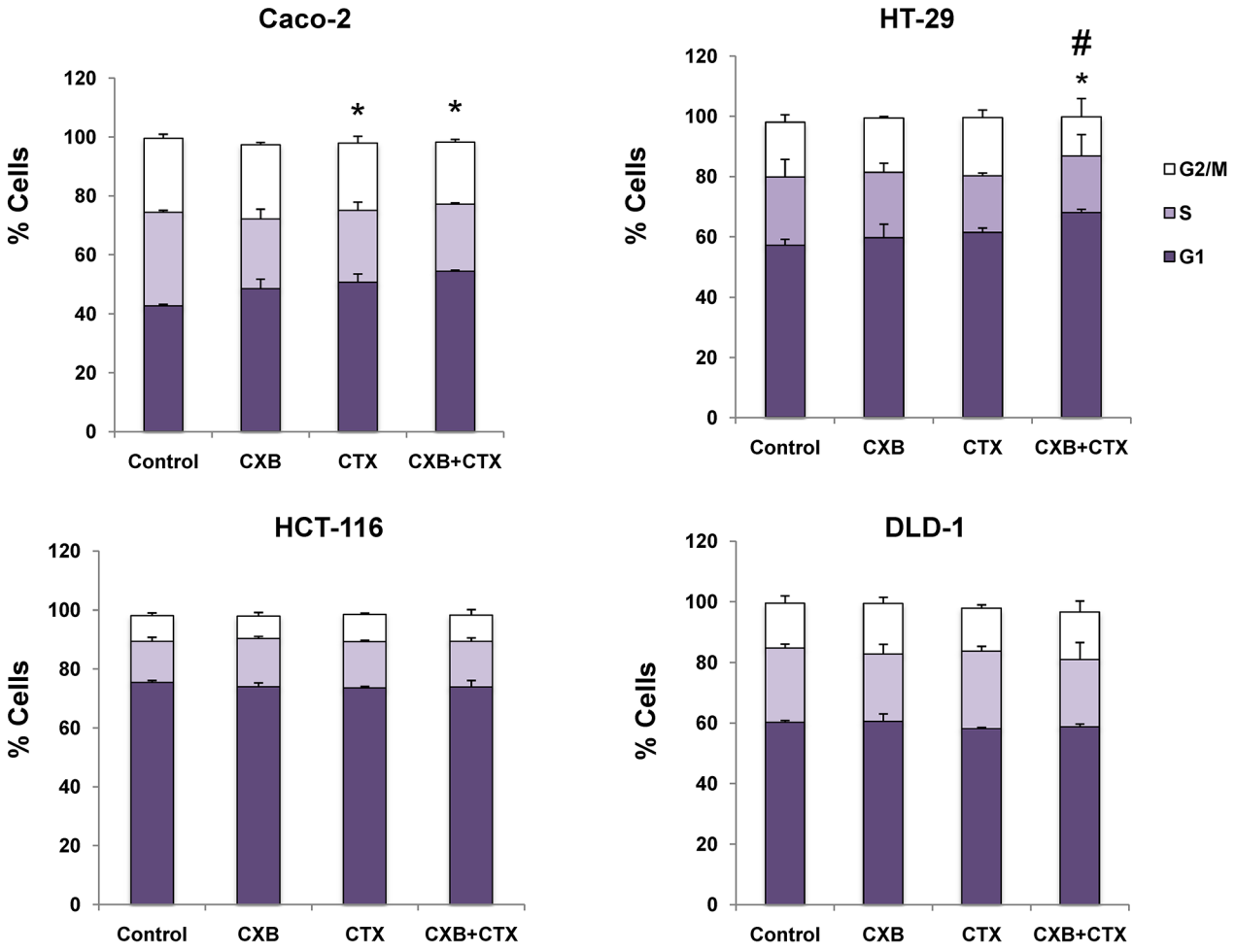
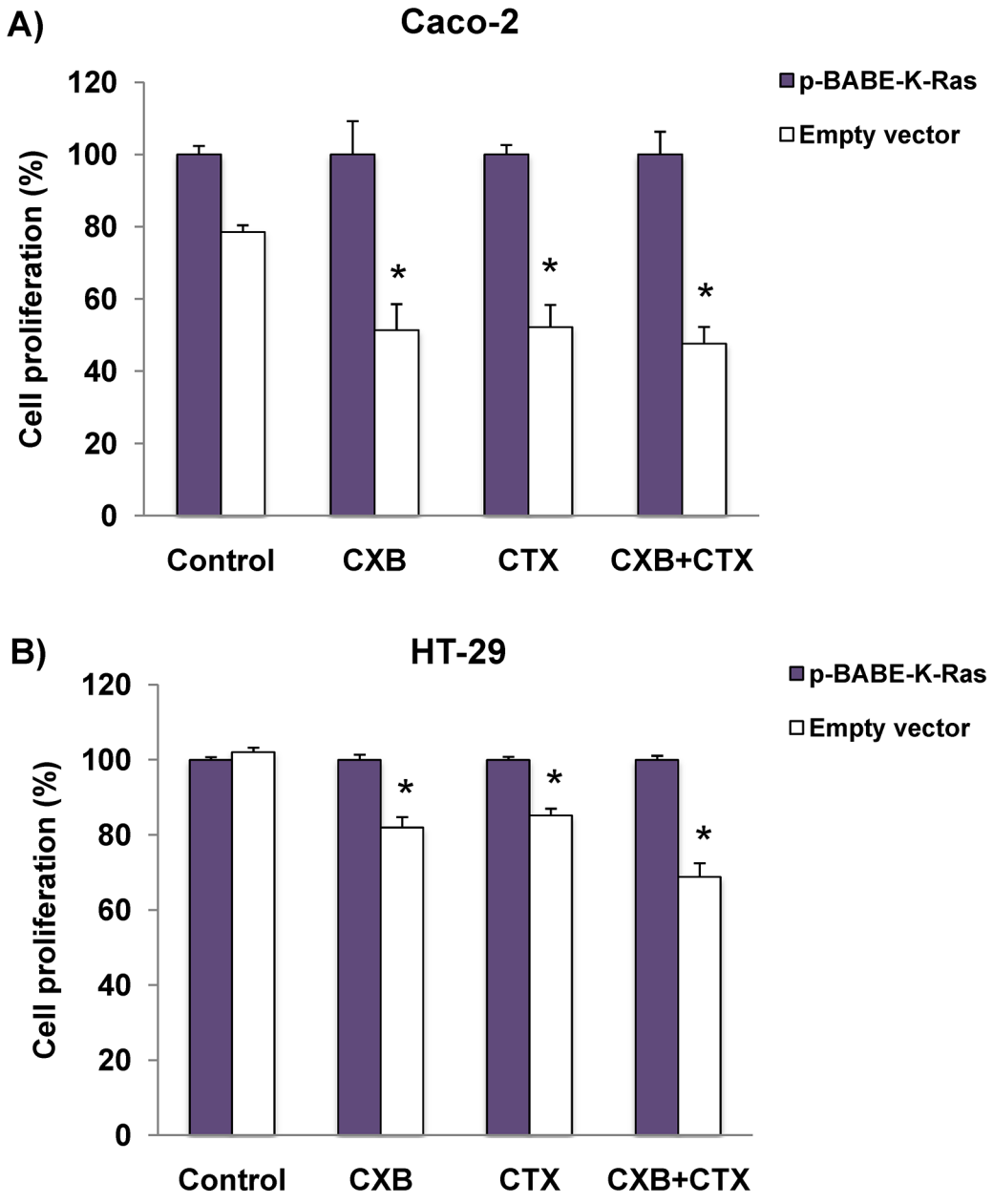
# The addition of celecoxib improves the antitumor effect of cetuximab in colorectal cancer: role of EGFR-RAS-FOXM1-β-catenin signaling axis

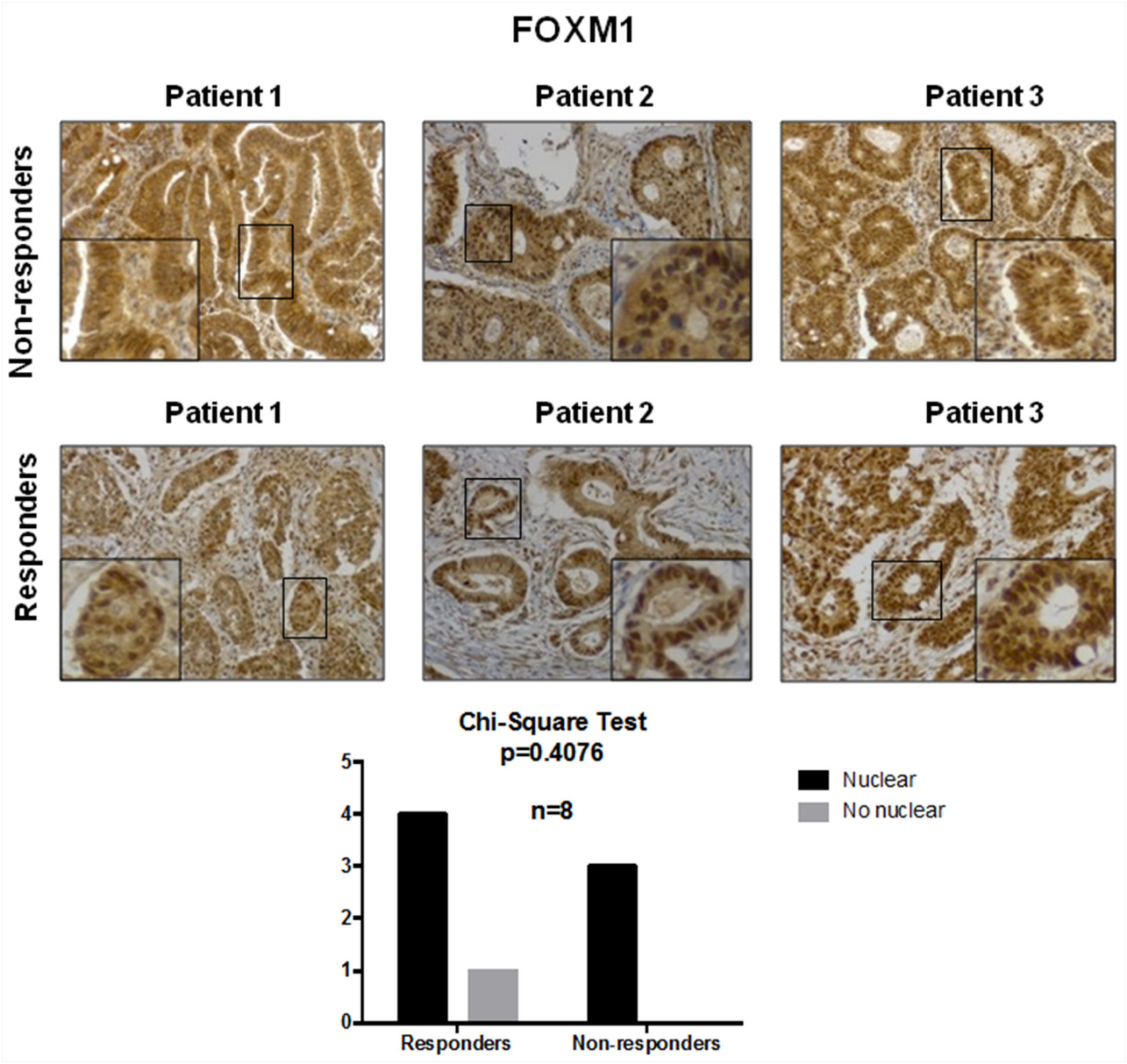
## SUPPLEMENTARY FIGURES




**Supplementary Figure 1: The combination of cetuximab with celecoxib alters cell cycle in colorectal cancer cells.** Cell cycle analysis was performed by flow cytometry after 48 h of treatment with cetuximab (CTX, 100 μg/ml) and/or celecoxib (CXB, 50 μM) of cells growing in the presence of EGF (100 ng/ml). Data are means ± SEM of three independent experiments (*p <0.05, compared with the control; # p<0.05, compared with cetuximab-treated cells).




**Supplementary Figure 2: Transient expression of mutant K-RAS gene in Caco-2 and HT-29 cells reduces the antiproliferative effects of the combination of cetuximab with celecoxib. A.** Caco-2 and **B.** HT-29 cells transiently transfected with pBabe K-Ras 12V plasmid showed reduced sensitivity to the antiproliferative effect of cetuximab and celecoxib. Data are means ± SEM of three independent experiments (*p <0.05, compared with the mutant K-RAS transfected cells).




**Supplementary Figure 3: FOXM1 expression in colon cancer and response of patients to the anti-angiogenic drug bevacizumab.** The expression of FOXM1 was analyzed by immunohistochemistry (IHC) in 8 human colorectal cancer tumors. **A.** Representative images of IHC stainings of tumors are shown. **B.** The association between the nuclear localization of FOXM1 and response to treatment was assessed by a chi-square test and *p* values were considered significant if they were < 0.05.