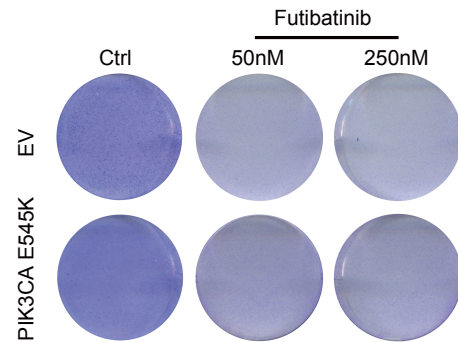
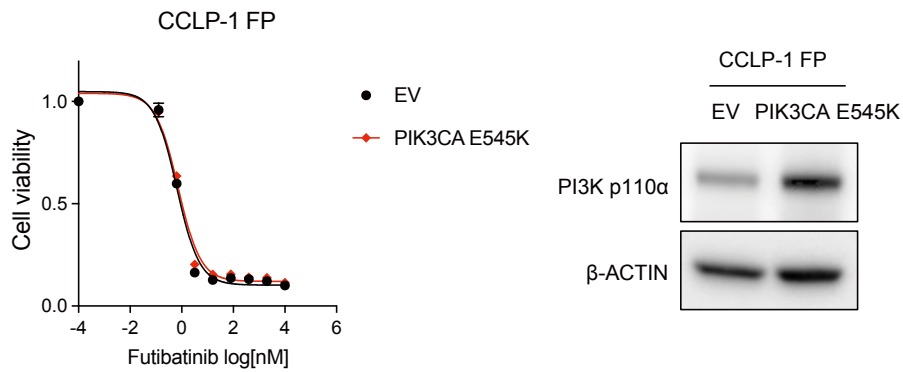
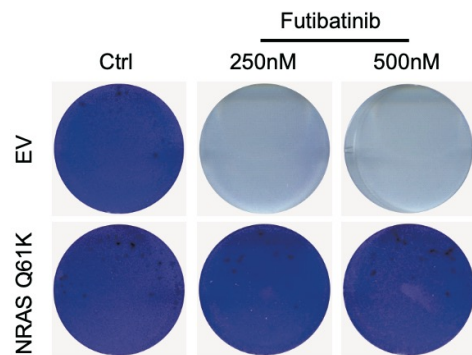
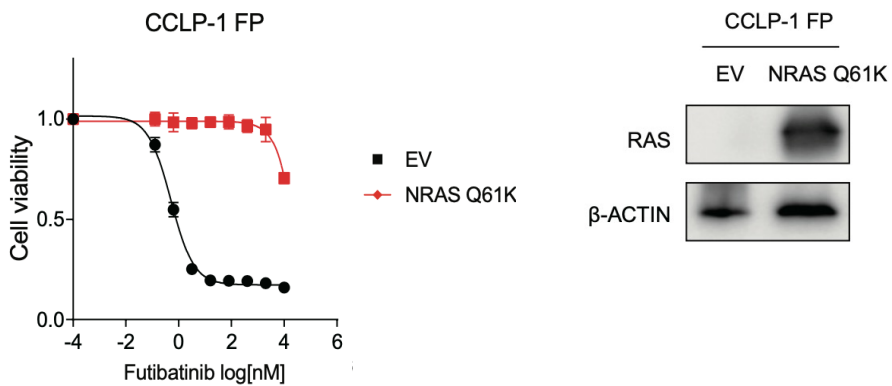


Supplementary Figure S1

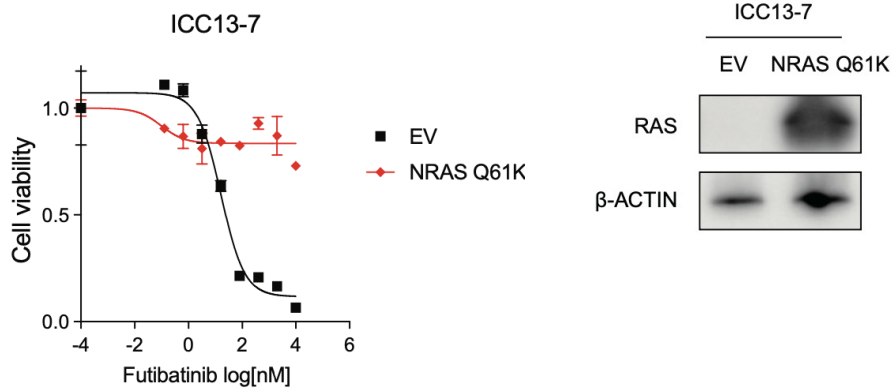
A



B



C



Supplementary Figure S1. RAS mutations drive resistance to FGFR inhibitors but not PIK3CA. A. The FGFR-dependent CCLP-1 FP cell line (harboring the FGFR2-PHGDN [FP] fusion) was engineered to stably express empty vector (EV) or PIK3CA-E545K. Cells were tested by IC50 assay for futibatinib sensitivity (left panel), immunoblot for PIK3CA (PI3K p110 α) (middle panel), and crystal violet staining under the indicated conditions (right panel). B-C. The FGFR-dependent CCLP-1 FP (B) and ICC13-7 cell lines (C) were engineered to stably express empty vector (EV) or NRAS-Q61K. IC50 assay for futibatinib sensitivity (left panel). Immunoblot for NRAS expression (middle panel). Crystal violet staining (right panel).