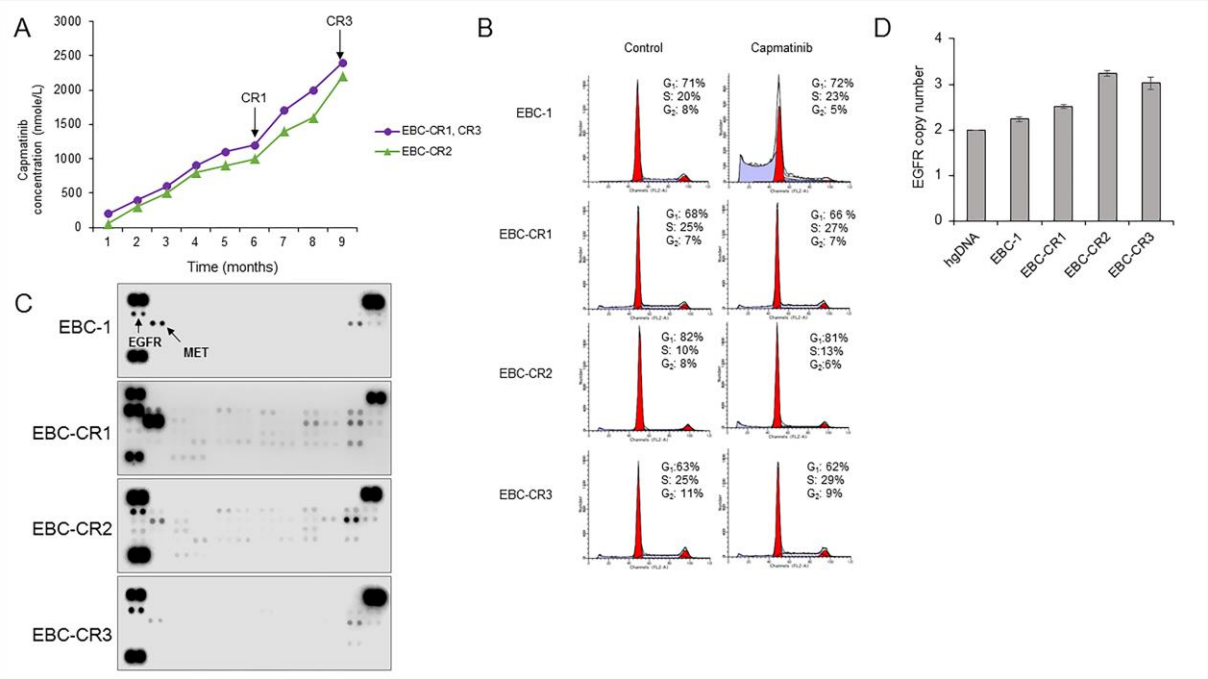

S3 Fig. Establishment of capmatinib-resistant cells from EBC-1. (A) EBC-1 cells were cultured in increasing concentrations of capmatinib from 10 nM to final concentrations of 1.5 (EBC-CR1), 2.2 (EBC-CR2), and 2.4 μmol/L (EBC-CR3) to establish capmatinib-resistant cell lines. The resistant cell lines derived from EBC-1 were cultured in increasing concentrations of capmatinib from 10 nmol/L to 2.4 μmol/L and were maintained at 1 μmol/L over 2 months. The EBC-CR3 cell line was derived from EBC-CR1 cells by treatment with a stepwise higher concentration of capmatinib over 3 additional months; this cell line had different molecular characteristics compared to the EBC-CR1 cell line. (B) Capmatinib significantly induced cell cycle arrest in sub-G1 phase on EBC-1. Cell cycle distribution of EBC-1 and capmatinib-resistant cells was analyzed by flow cytometry. (C) Epidermal growth factor receptor (*EGFR*) copy number was not altered in the resistant cell lines. *EGFR* copy number was confirmed by quantitative polymerase chain reaction. (D) EGFR phosphorylation was increased in all resistant cell lines, especially in EBC-CR1. Human phospho–receptor tyrosine kinase (RTK) arrays were used to compare the activation of multiple RTKs between parental and resistant cell lines.