



Supplementary Figure 5: B-I09 is not effective against *ARID1A* wildtype OCCCs. (A) Schematic of experimental design for orthotopic xenograft mouse model using OCCC cell lines. (B-E) Orthotopic xenografts formed by *ARID1A* wildtype RMG1 cells treated with vehicle or B-I09. Shown are images of reproductive tracks with tumors from indicated groups (B). Tumor weight was measured as surrogate for tumor burden (n = 10 mice for vehicle treated group and n = 9 mice for B-I09 treated group) (C). Dissected tumors from the indicated treatment groups were subjected to immunohistochemical (IHC) staining for the cell proliferation marker Ki67 or the apoptotic marker cleaved caspase 3 on serial sections (D), and the histological scores (H-score) of indicated markers were quantified in each of the indicated treatment groups (E). Scale bar = 100 μ m. (F) Schematic of experimental design for orthotopic patient-derived xenograft for *ARID1A*-mutant OCCCs. (G) Loss of ARID1A protein expression was confirmed by IHC staining in *ARID1A*-mutated OCCC PDX that carries the I832fs frameshift mutation. Staining of tumors formed by *ARID1A* wildtype RMG1 cells were used as a positive control for ARID1A staining. (H) Schematic of experimental design for the genetic *Arid1a*^{-/-}/*Pik3ca*^{H1047R} OCCC mouse model used for assessing the efficacy of B-I09 treatment. (I) Body weight of tumor bearing mice were measured at the indicated time point during B-I09 treatment in the genetic *Arid1a*^{-/-}/*Pik3ca*^{H1047R} OCCC mouse model (n = 5 mice per group). Scale bar = 100 μ m. *P* values were calculated using two-tailed Student's *t*-test. Error bars represent mean with SD.