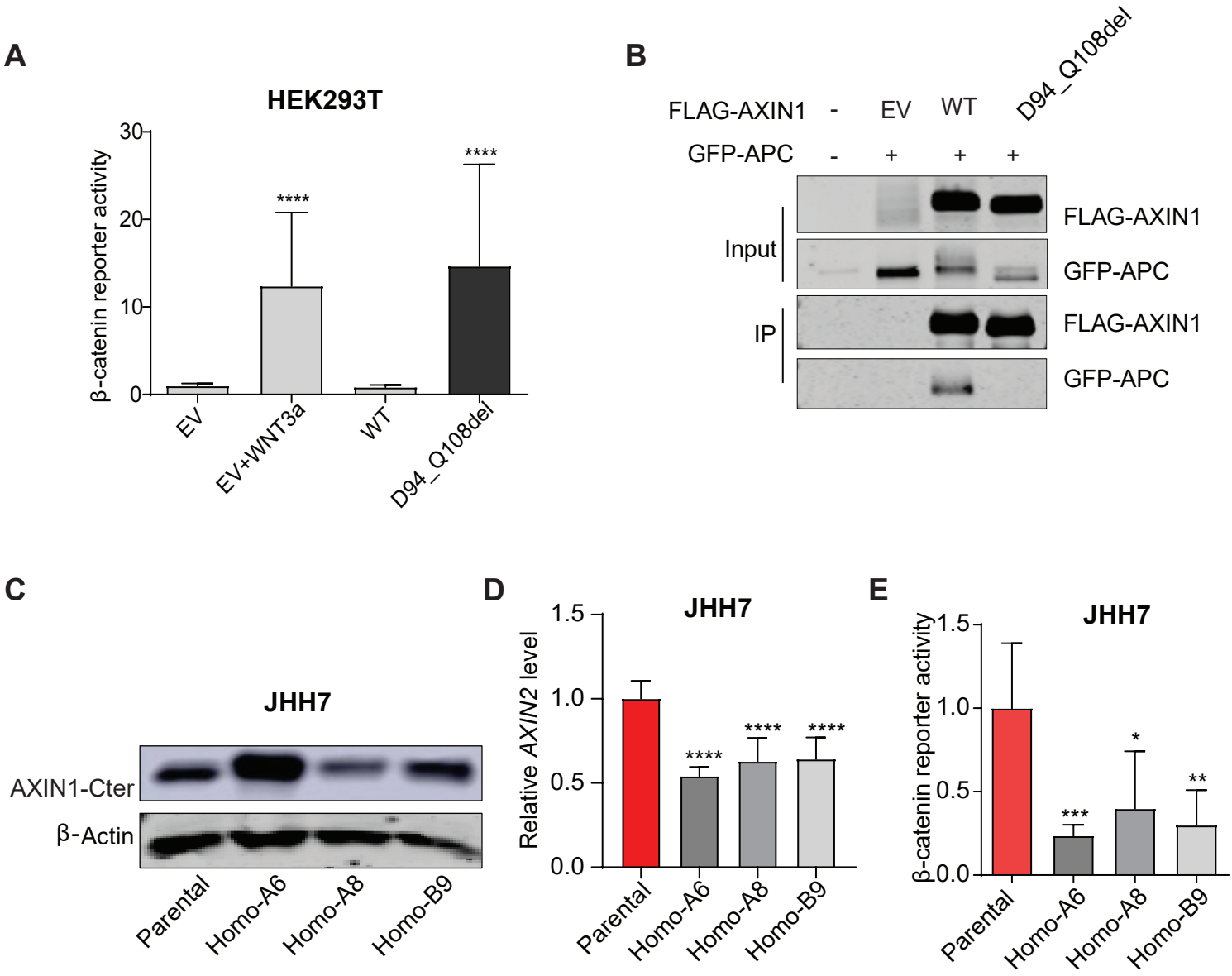

Supplementary Fig.11 Deleting part of the RGS/APC domain at endogenous levels results in increased β-catenin signaling. To this aim, the homozygous D94_Q108del deletion present in JHH7 liver cancer cells was corrected using gene editing. (A) A β-catenin reporter assay shows that expression of the D94_Q108 variant strongly increases signaling, and (B) is unable to co-precipitate GFP-APC. (C) AXIN1 immunoblot of three independent JHH7 clones obtained through Crispr-Cas9 and HDR mediated repair of the original mutation. (D) QRT-PCR assay showing that *AXIN2* mRNA levels are significantly reduced in the AXIN1-repaired JHH7 clones (in triplicate, n=2 independent experiments). Expression levels are relative to the housekeeping gene *GAPDH.* The value for the wild-type control is arbitrarily set to 1. (E) Likewise, a β-catenin reporter assay shows lower levels of signaling in all three repaired clones (in triplicate, n=2 independent experiments). All data shown as mean ± SD. Statistical significance for all experiments was analyzed using a Mann-Whitney test (*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$, ****$P < 0.0001$).