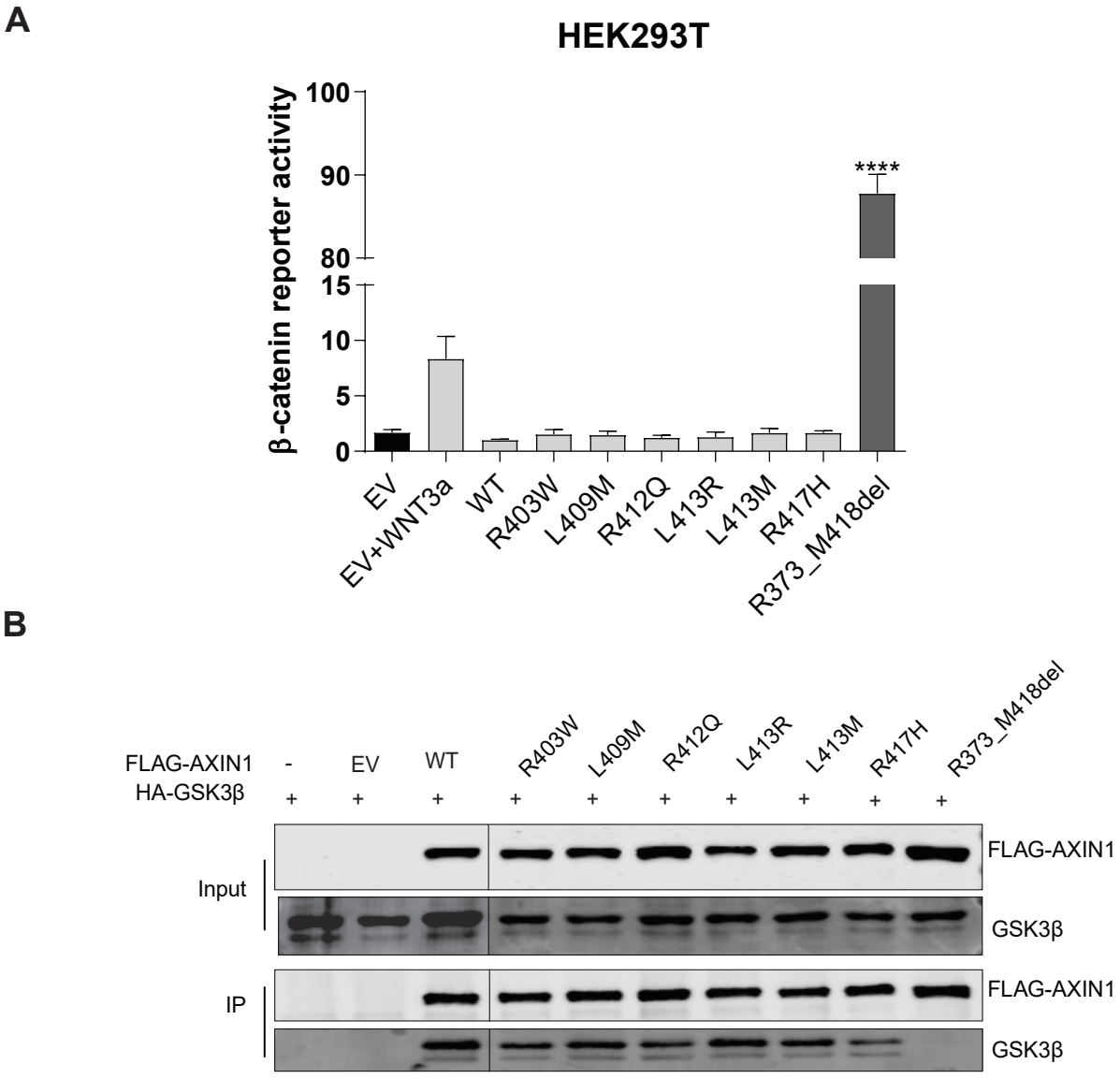

Supplementary Fig.7 Six additional tumor-associated AXIN1 variants observed at conserved residues directly following the GSK3β domain, were investigated for their ability to affect β-catenin signaling and GSK3β binding. (A) A β-catenin reporter assay shows that all six additional missense variants still preserve the ability to regulate β-catenin levels. Wild-type AXIN1 (WT) and the R373_M418del variant were included, respectively, as positive and negative controls. The P385A and L396M variants were also included in Figure 4. (B) All variants are capable to efficiently co-precipitate endogenous (lower band) and co-transfected HA-GSK3β, in contrast with the R373_M418del variant. Two irrelevant lanes have been removed from original blots.