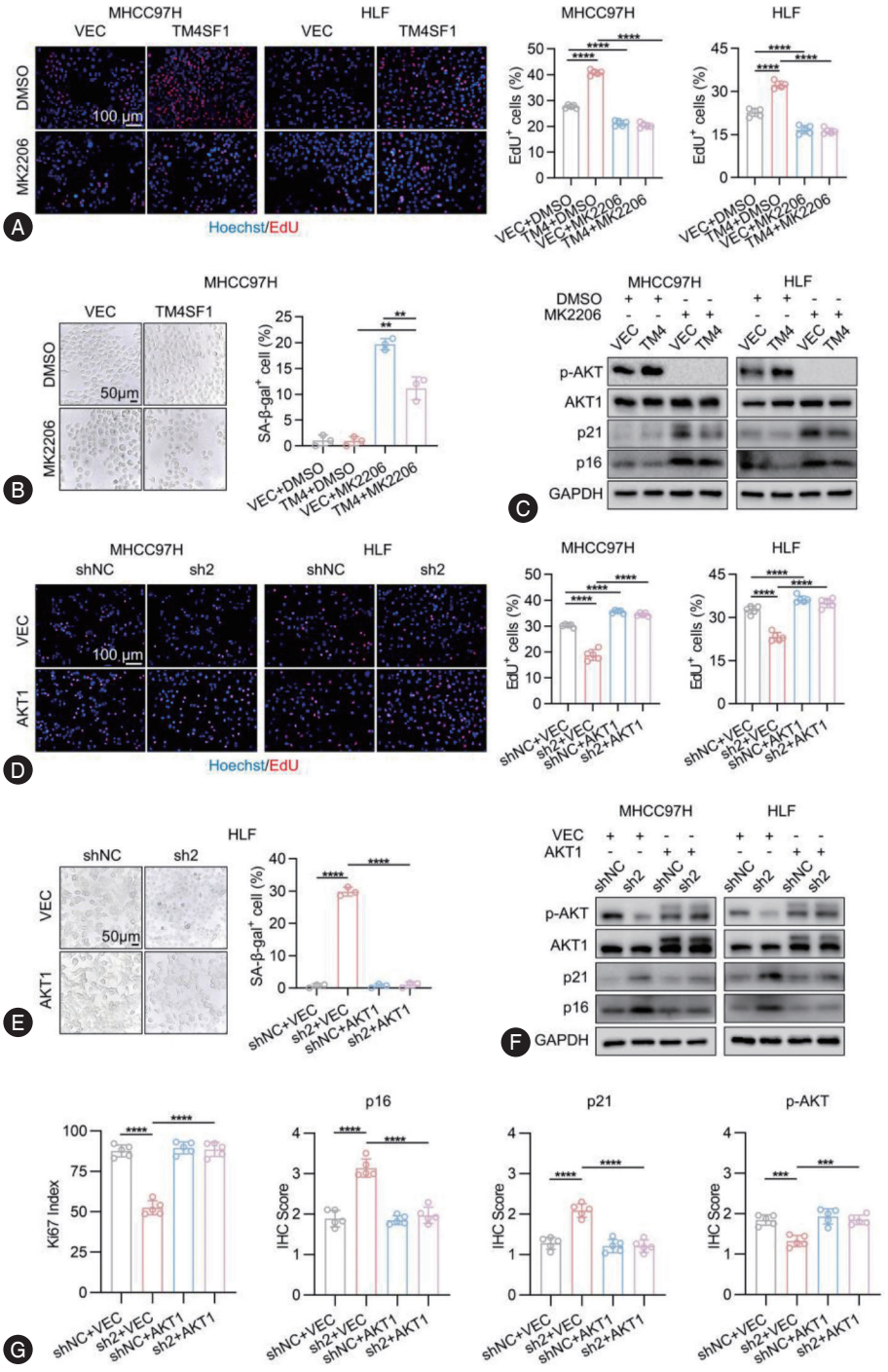

Supplementary Figure 5. TM4SF1 promotes AKT phosphorylation. (A) EdU assay in MHCC97H and HLF which were treated with MK2206 (150 nM) for 24 hours on the basis of TM4SF1 overexpression, with statistical analysis. (B) SA-β-gal staining in MHCC97H which were treated with MK2206 (150 nM) for 24 hours on the basis of TM4SF1 overexpression, with statistical analysis. (C) Expression levels of p-AKT-S473, p21 and p16 in MHCC97H and HLF which were treated with MK2206 (150 nM) for 24 hours on the basis of TM4SF1 overexpression. (D) EdU assay in MHCC97H and HLF with AKT1 supplementation on the basis of TM4SF1 knockdown, with statistical analysis. (E) SA-β-gal staining in HLF with AKT1 supplementation on the basis of TM4SF1 knockdown, with statistical analysis. (F) Expression levels of p-AKT-S473, p21 and p16 in MHCC97H and HLF with AKT1 supplementation on the basis of TM4SF1 knockdown. (G) IHC intensity of orthotopic tumor model with AKT1 supplementation (n=5). Data are represented as means±standard deviation in the bar graphs. *P*-values were calculated using the Student's *t*-test (unpaired). **P*<0.05, ***P*<0.01, ****P*<0.001, *****P*<0.0001. TM4SF1, transmembrane 4 L six family member 1; SA-β-gal, senescence-associated beta-galactosidase; IHC, immunohistochemistry.

 https://doi.org/10.3350/cmh.2024.0934