



Correction: A novel UBE2T inhibitor suppresses Wnt/ β -catenin signaling hyperactivation and gastric cancer progression by blocking RACK1 ubiquitination

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Correction to: *Oncogene*

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The original version of this article unfortunately contained a mistake. In Fig. 6D, the abscissa should be “concentration [μ M]” and in the figure caption of Fig. 6D, the concentration range should be given as “31 nM to 500 μ M”. The corrected figure and figure caption is given below. The original article has been corrected.

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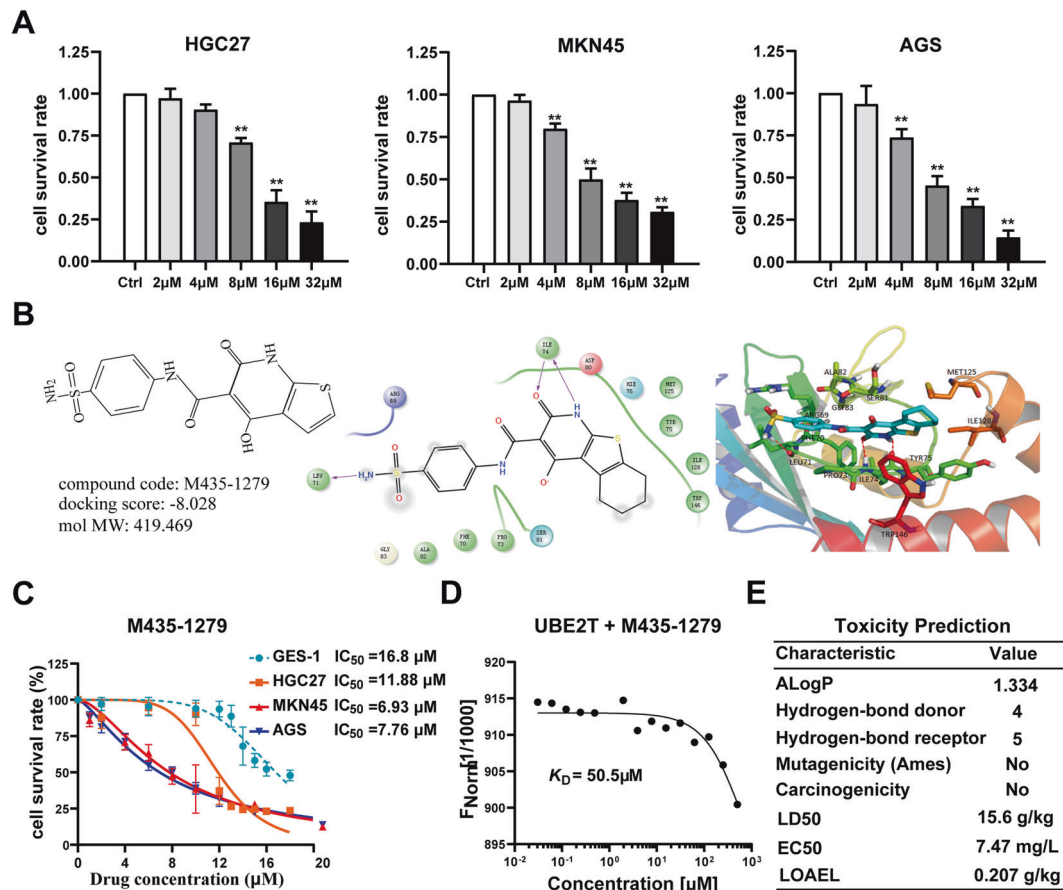


Fig. 6 Identification of UBE2T inhibitor. **A** Cell survival rate of HGC27, AGS, and MKN45 cells after 48 h treated with M435-1279. Cell viability was detected by 3-(4,5-dimethyl-2-thiazolyl)-2,5-diphenyl-2-H-tetrazolium bromide (MTT) assay. One-way analysis of variance (ANOVA) was used to examine statistical significance (mean \pm S.D., $n = 6$, $**P < 0.01$, $*P < 0.05$). **B** A simulation snapshot of M435-1279 with the allosteric site of UBE2T by molecular dynamics simulations. Sticks defined as the compounds and the active sites interacted with UBE2T, red thread dotted lines defined as hydrogen bonds between the compounds and UBE2T. For the compound, hydrogen: white, carbon: blue, oxygen: red, nitrogen: dark blue, and sulfur: yellow. **C** The effect of M435-1279 on the growth of HGC27, AGS, MKN45, and GES-1 (as control), respectively. Cell viability was detected by 3-(4,5-dimethyl-2-thiazolyl)-2,5-diphenyl-2-H-tetrazolium bromide (MTT) assay. **D** The binding of fluorescently labeled UBE2T to M435-1279 is analyzed with microscale thermophoresis (MST) assay. The M435-1279 is titrated from 31 nM to 500 μ M. The change in the thermophoretic signal leads to a $K_D = 50.5 \mu$ M. **E** The toxicity of M435-1279 is predicted based on TOPKAT analysis. ALogP lipophilicity (< 5 value shows good lipophilicity), LD50 50% lethal dose of a chemical that kills 50% of a sample population, EC50 water flea EC50, 50% effective concentration, LOAEL lowest-observed-adverse-effect level.