

CORRECTION

Correction to: Inhibition of p53 and/or AKT as a new therapeutic approach specifically targeting ALT cancers

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In the original publication the labels in Fig. 4C and 4D are incorrectly published. The correct labels for Fig. 4C and 4D is provided in this correction.

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Yuanlong Ge and Shu Wu have contributed equally to this work.

The original article can be found online at <https://doi.org/10.1007/s13238-019-0634-z>.

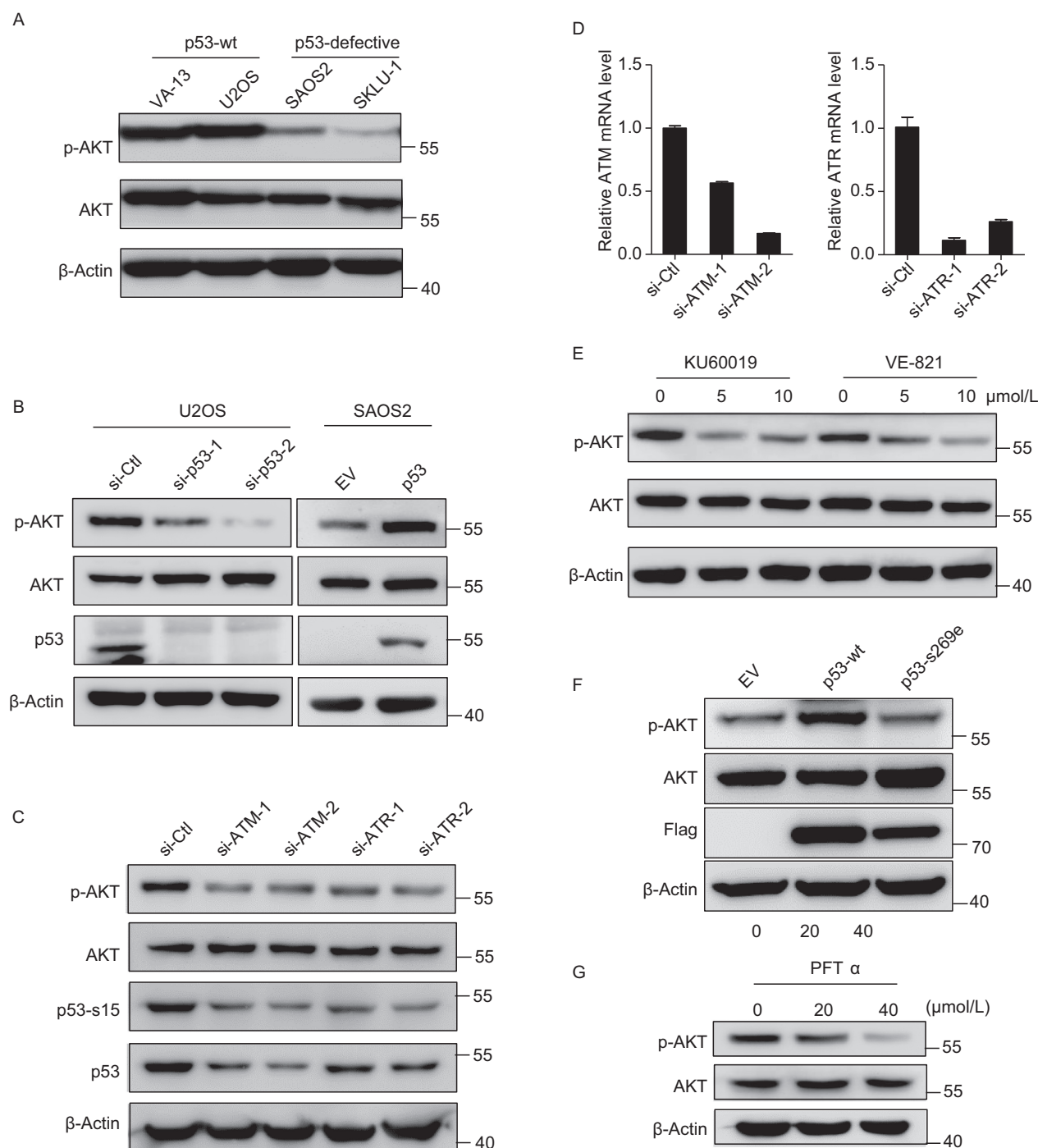


Figure 4. AKT is phosphorylated in p53-dependent manner in ALT cells. (A) Western blot determination of total and phosphorylated AKT (S473) in p53-positive (VA13, U2OS) and p53-defective (SAOS2, SKLU-1) ALT cells. (B) Knockdown of p53 in U2OS or moderate expression of p53 in SAOS2 induces down or up-regulation of p-AKT, respectively. (C) Knockdown of ATM or ATR by siRNA decreases abundance of p53, phosphorylated p53 and p-AKT. (D) Quantitative-PCR determination of the level of ATR or ATM in U2OS cells transfected with siRNA to ATR or ATM, respectively. Scramble siRNA (Si-Ctl) was used as control. Data represent the mean $\pm$ SEM, n=3-4. (E) ATM (KU60019) or ATR (VE-821) inhibitor decreases abundance of p-AKT in U2OS cells. U2OS cells were treated with indicated concentration of KU60019 or VE-821 for 24 h. (F) The expression of wt-p53, but not mutant p53 (p53-s269e) defective of transcription activity, increases the level of p-AKT. (G) PFT α , an inhibitor of p53 transcription activity, suppresses the phosphorylation of AKT. U2OS cells were treated with indicated concentration of PFT α for 24 h.