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CORRECTION

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

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## CORRECTION TO: PROTEIN CELL HTTPS://DOI.ORG/10.1007/S13238-019-0634-Z

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.



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.

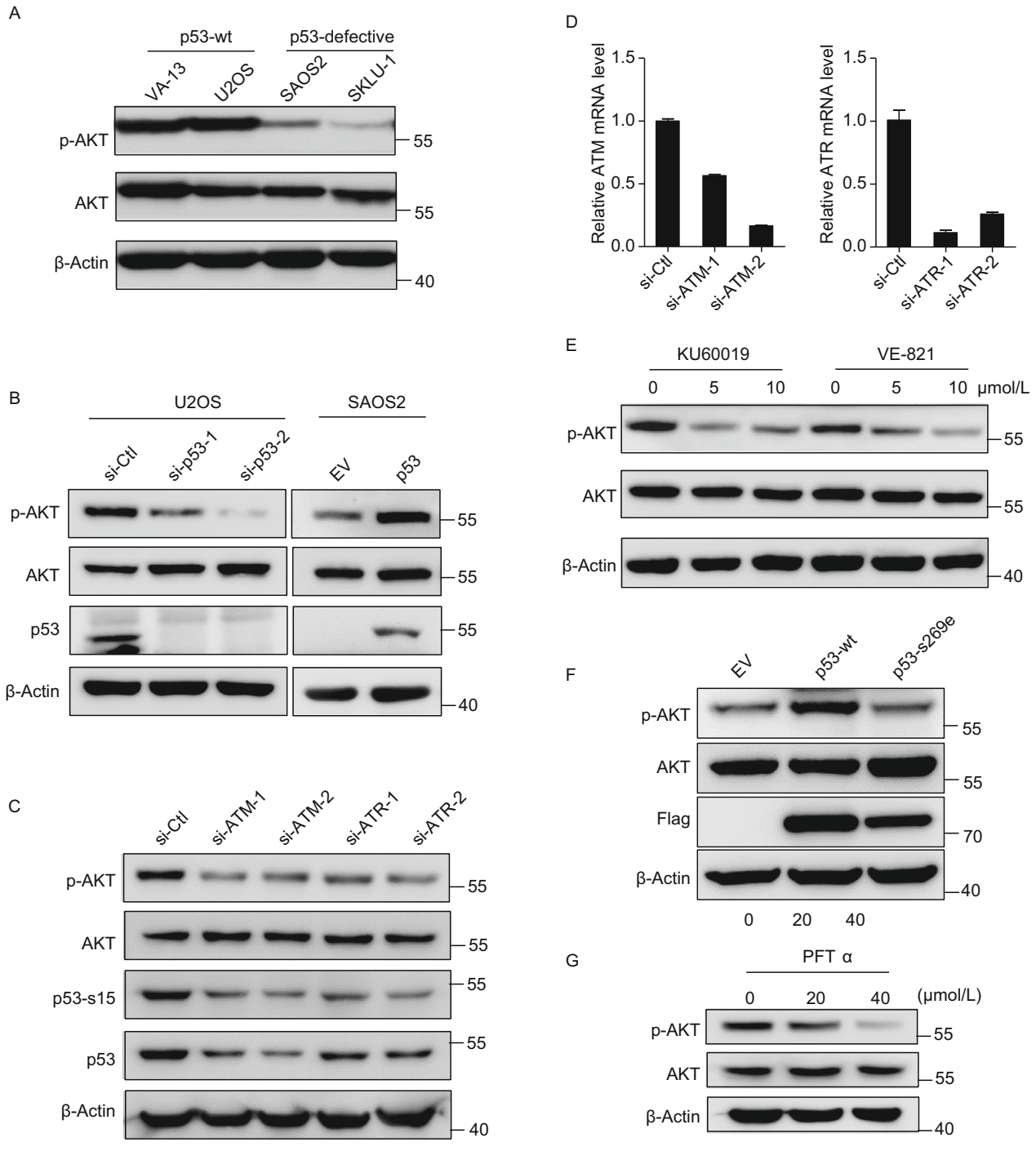


**Figure 4. AKT is phosphorylated in p53-dependent manner in ALT cells.** (A) Western blot determination of total and phosphorylated AKT (S473) in p53-positive (VA13, U2OS) and p53-defective (SAOS2, SKLU-1) ALT cells. (B) Knockdown of p53 in U2OS or moderate expression of p53 in SAOS2 induces down or 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±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) PFTα,an inhibitor of p53 transcription activity, suppresses the phosphorylation of AKT. U2OS cells were treated with indicated concentration of PFTα for 24 h.