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# Author Correction: Characterisation of a cyclic peptide that binds to the RAS binding domain of phosphoinositide 3-kinase p110 $\alpha$

Published online: 13 March 2025

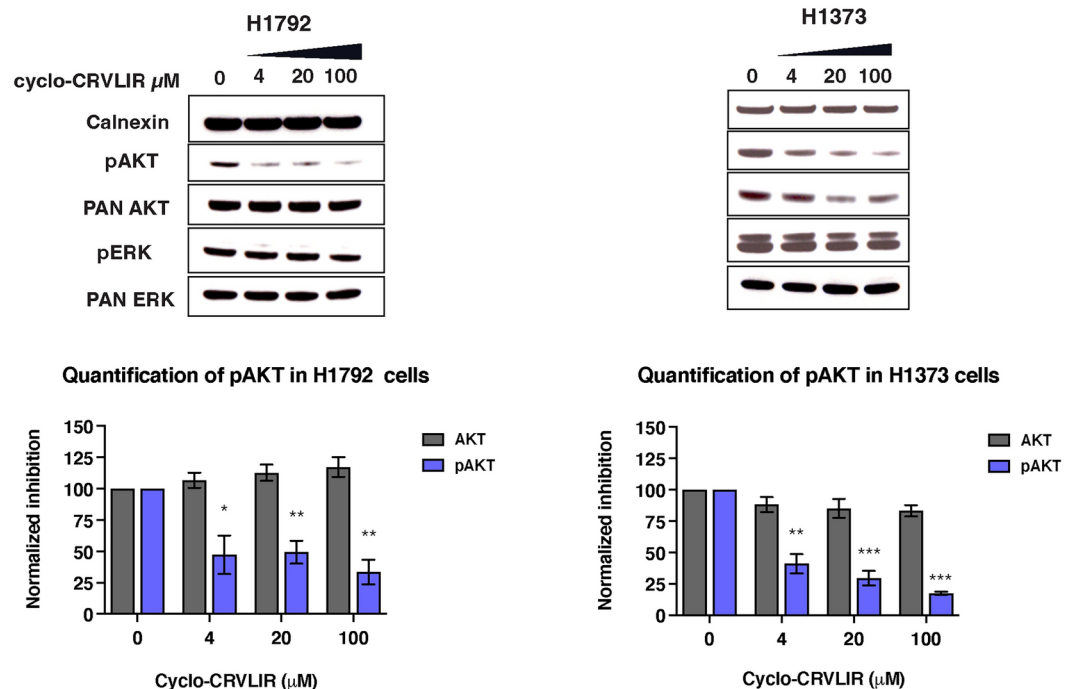
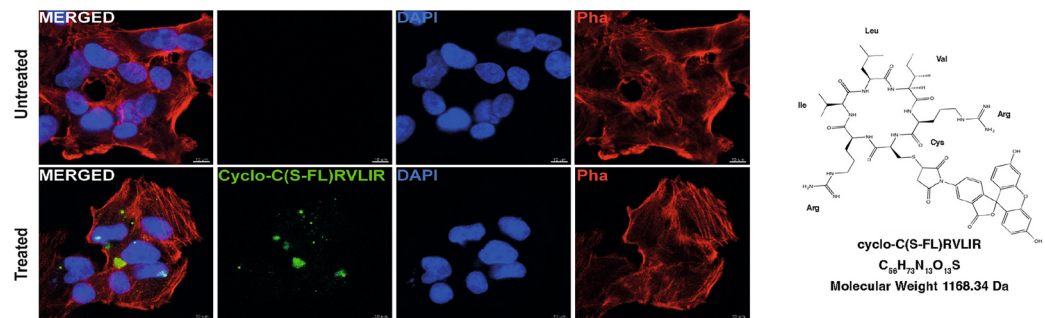
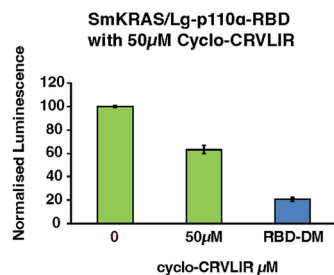
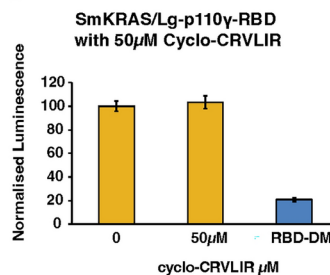
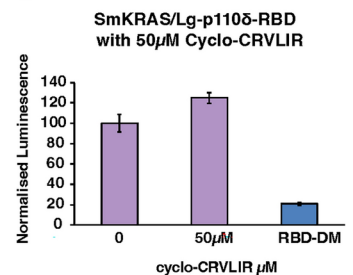
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Correction to: *Scientific reports* <https://doi.org/10.1038/s41598-023-28756-0>, published 02 February 2023

The original version of this Article contained an error.

As a result of an error during assembly of Fig. 3, the blot representing pan-AKT for H1373 (A) was duplicated from pan-ERK. The original Fig. 3 and accompanying legend appear below.

The original Article has been corrected.

**A****B****Cellular uptake of cyclo-C(S-FL)RVLIR in H1792 cells****C****D****E**

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© The Author(s) 2025 **Fig. 3.** Analysis of the effect of cyclo-CRVLIR in cancer cell lines and NBBA. (A) H1792 and H1373 cells were treated with increasing concentrations of cyclo-CRVLIR (4, 20 and 100  $\mu\text{M}$ ) for 4 h. Cell lysates were probed with the indicated antibodies. Bottom graphs show expression of phospho-AKT

(anti-pAKT-S473) and total AKT (normalised to calnexin expression). Mean  $\pm$  SEM, N = 3, un-paired Student's t-test treated vs untreated cells. Original blots with multiple exposure times are presented in Supplementary Fig. 6 with the main blot presented in Fig. 3A red box. **(B)** Cellular uptake of the fluorescein-conjugated cyclo-C(S-FL)RVLIR in H1792 cells. Representative images of H1792 cells, stained for DAPI (blue) and Phalloidin (red), after treatment with 100  $\mu$ M of the peptide (green) for 24 h, on the right is the structure of the fluorescein-conjugated C(S-FL)RVLIR. **(C–E)** Testing the specificity of Cyclo-CRVLIR to RBD $\alpha$  using the NBBA. The three RAS binding domains of PI3K isoforms (Lg-RBD $\alpha$ , Lg-RBD  $\delta$  and Lg-RBD $\gamma$ ) were transfected with Sm-KRAS in HEK293 cells, and cell lysates were treated with 50  $\mu$ M cyclo-CRVLIR. Only Sm-KRAS/Lg-RBD $\alpha$  showed reduction in the interaction signal and not the other RBDs, demonstrating that cyclo-CRVLIR is an RBD $\alpha$  specific peptide. RBD-DM (a p110 $\alpha$ -RBD with two mutations, T208D and K227A, that does not bind to RAS) was cloned and expressed in the Lg-BiT (Lg-RBD-DM). In the control experiments, Sm-KRAS-G12C was co-transfected with Lg-RBD-DM and the lysate was used as a negative control to indicate the true signal reduction upon the inhibition of the RAS/p110 $\alpha$  interaction.