

Loss of RET promotes mesenchymal identity of neuroblastoma cells

Original images

Fig 1-A

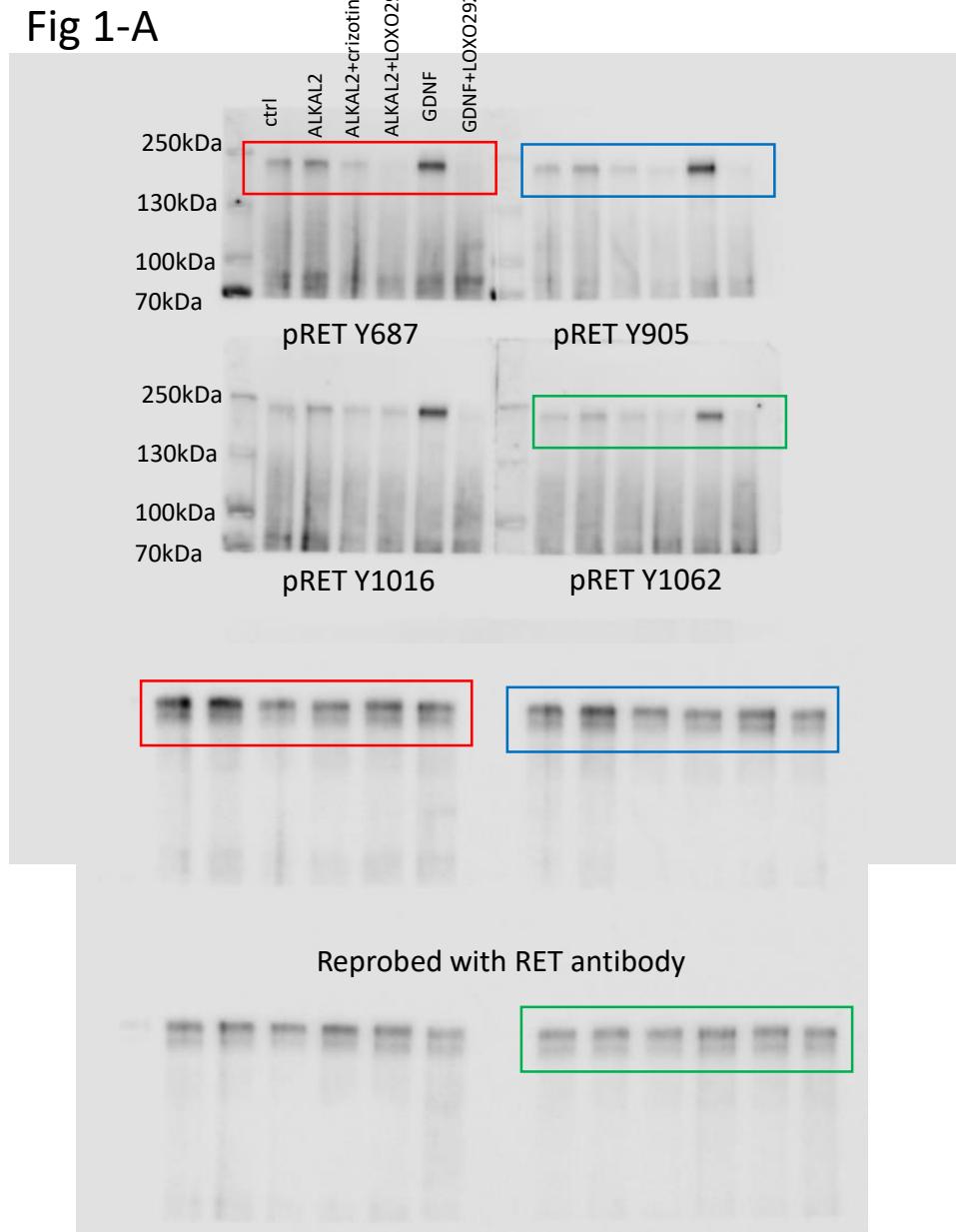


Fig 1-B

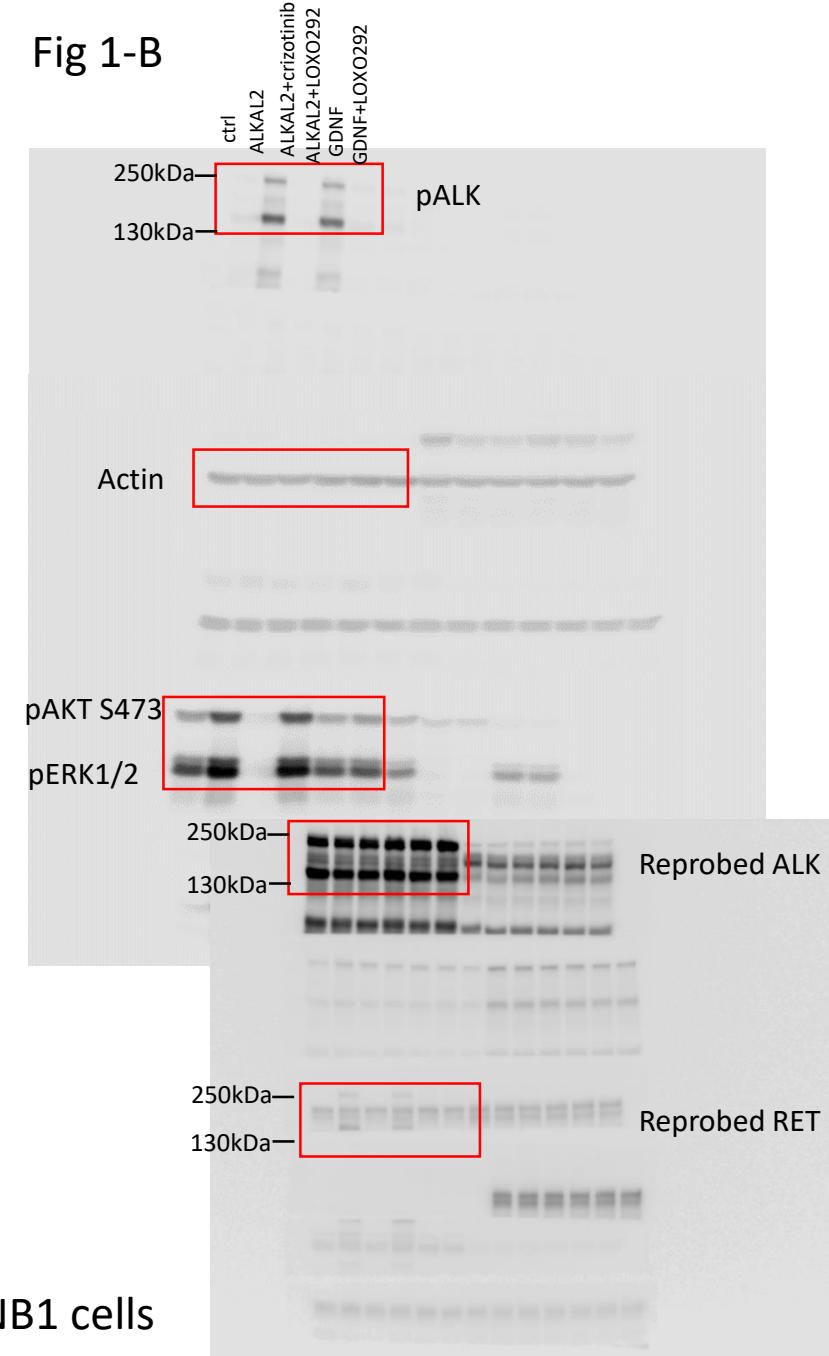


Fig 1C

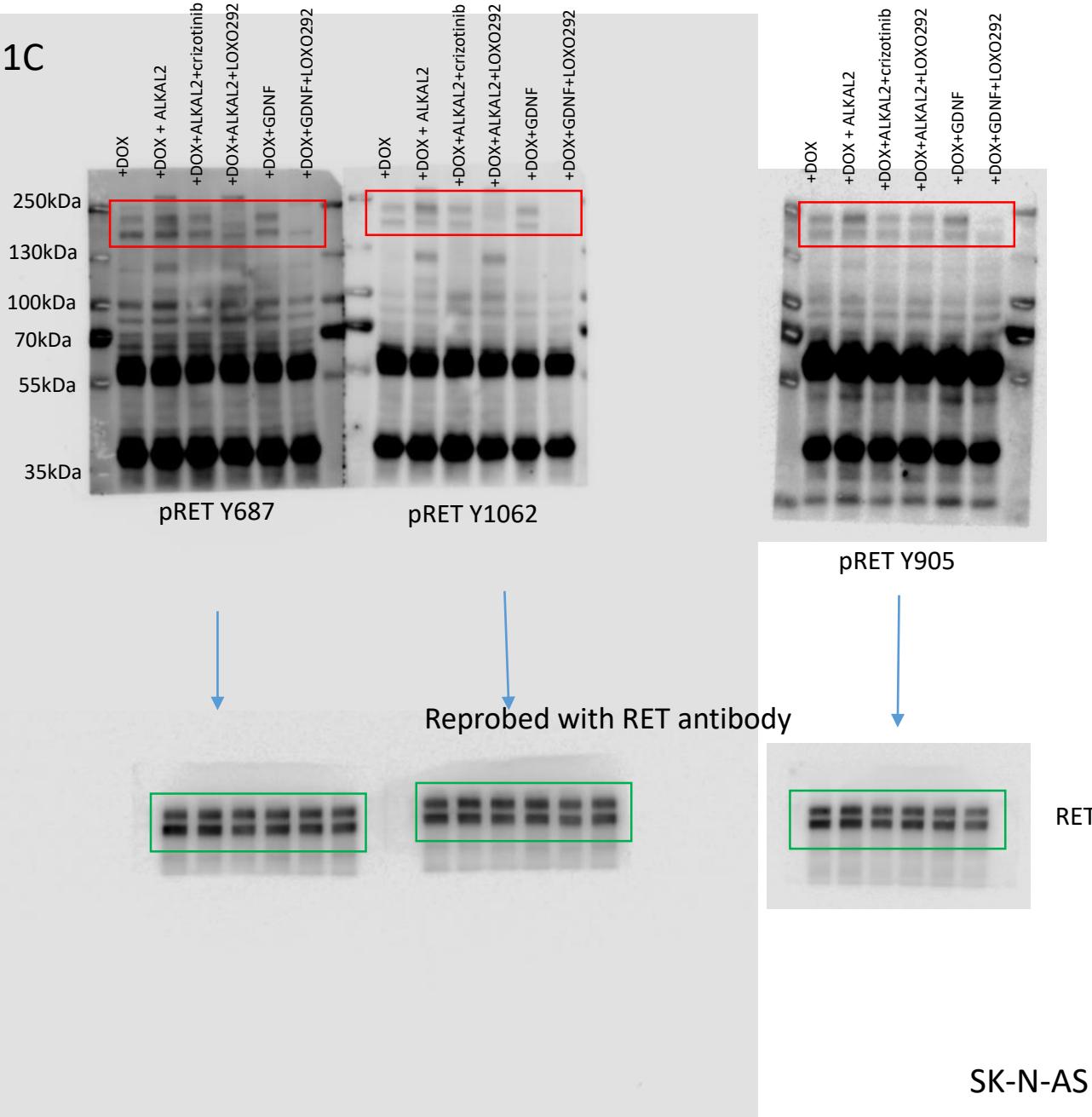


Fig 1D

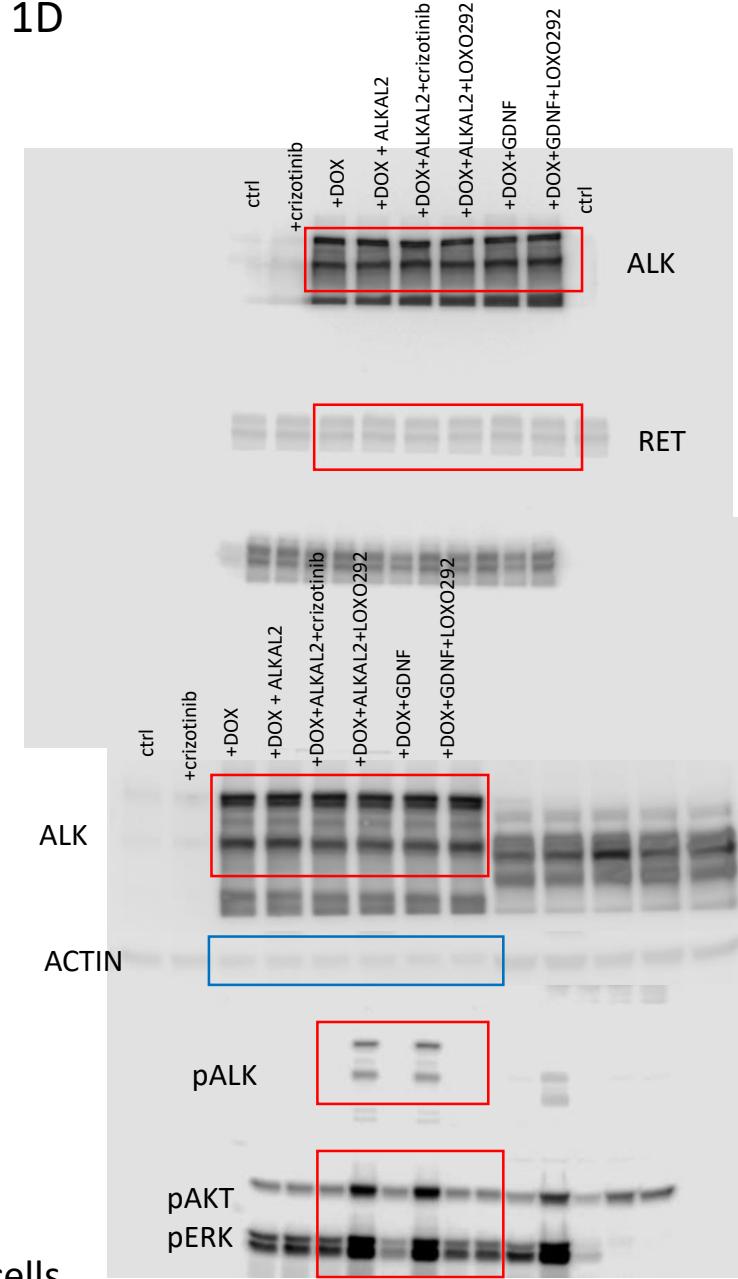


Fig. 2A-B ALK IP RET in NB1 cells.

Fig. 2A

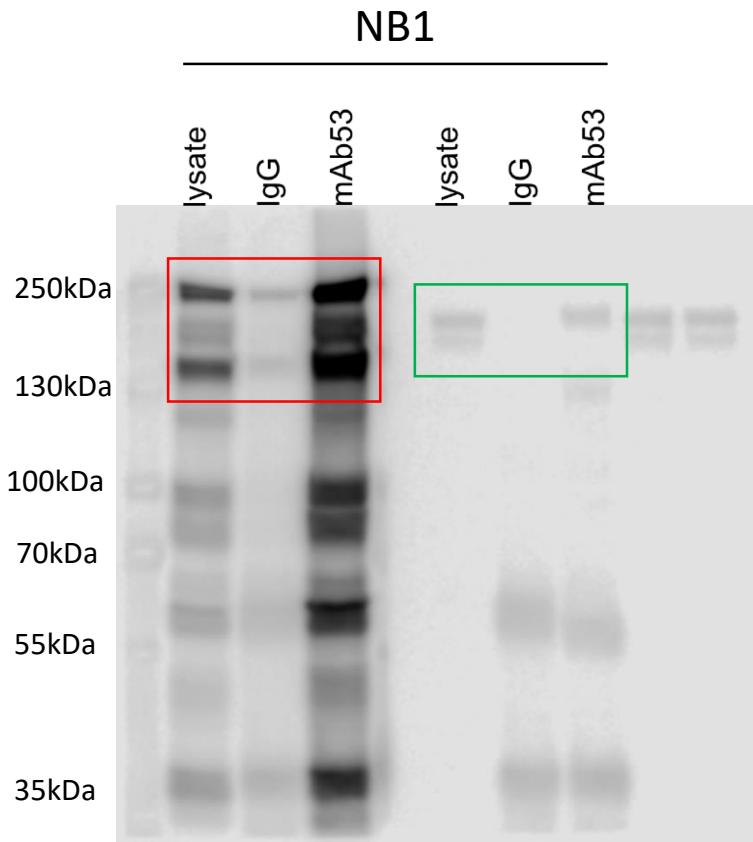
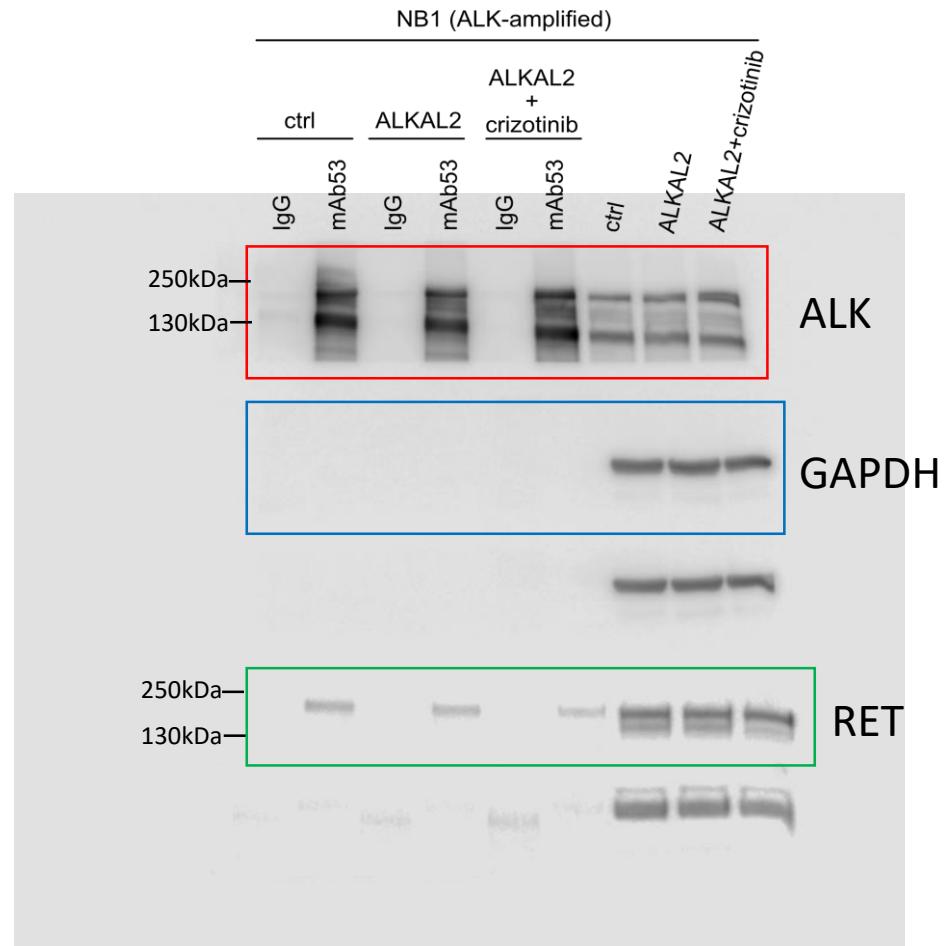
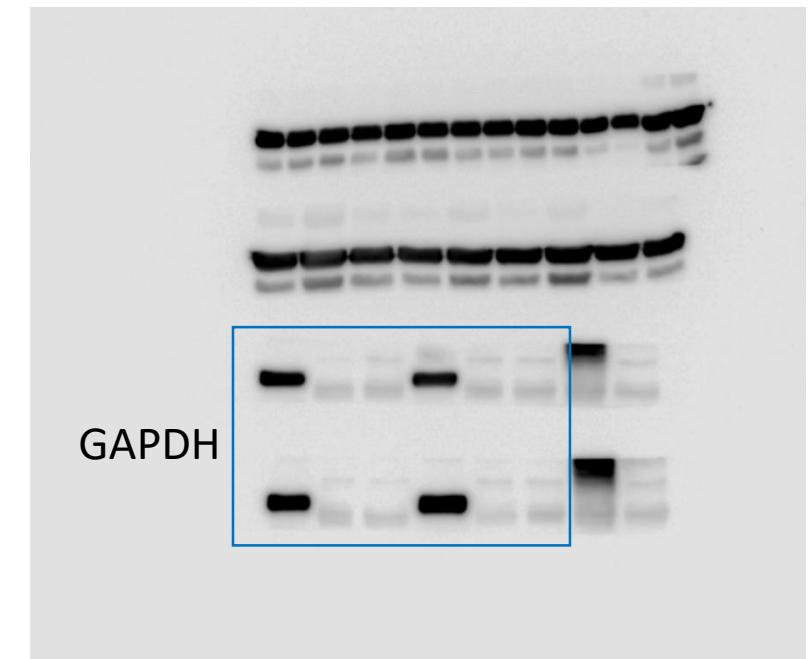
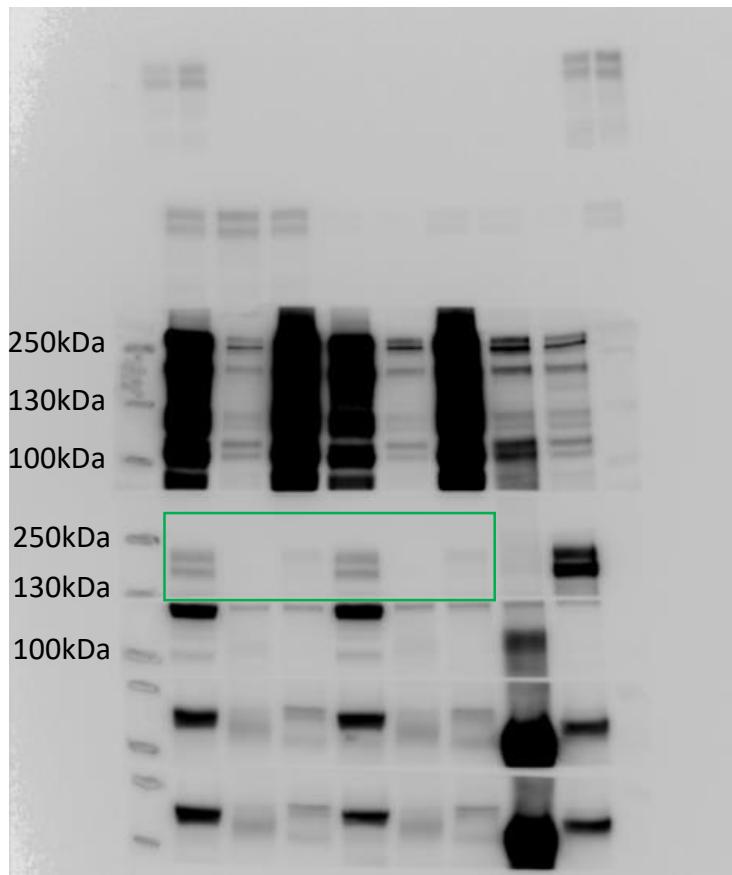
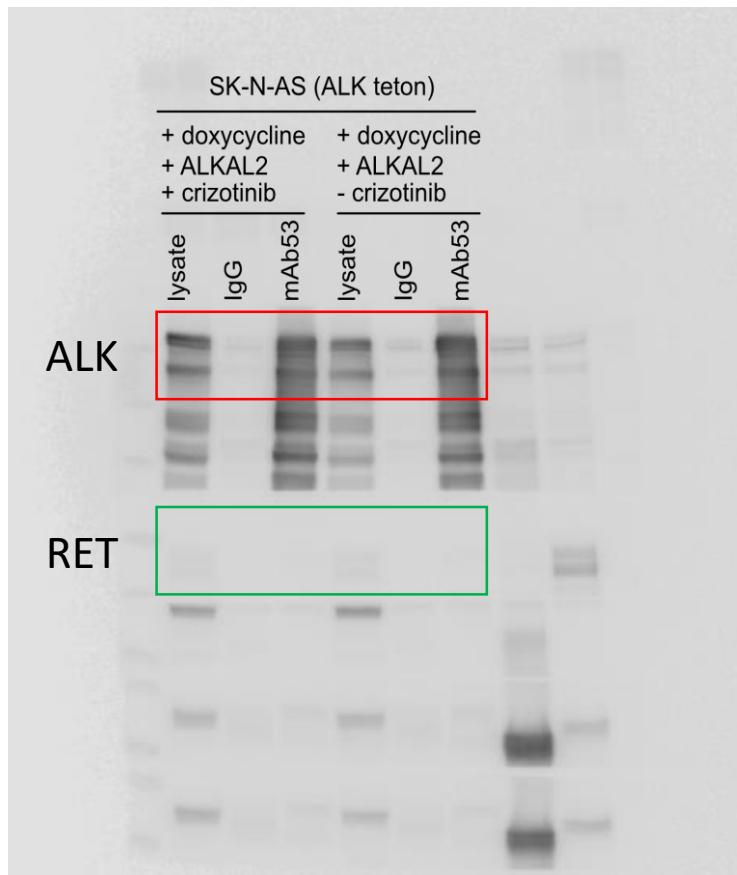


Fig. 2B



Red box: ALK blot used in main figure
Green box: RET blot
Blue box: GAPDH control

Fig. 2C-ALK IP RET in SK-N-AS (ALK teton) cells



Red box: ALK blot used in main figure
Green box: RET blot
Blue box: GAPDH control

Fig 3A

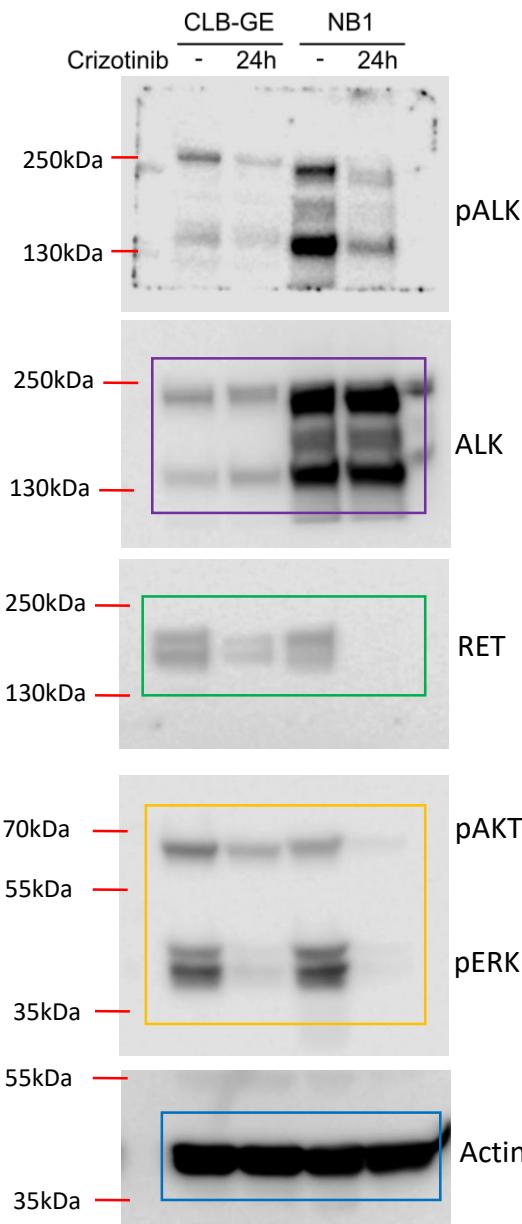


Fig 3B

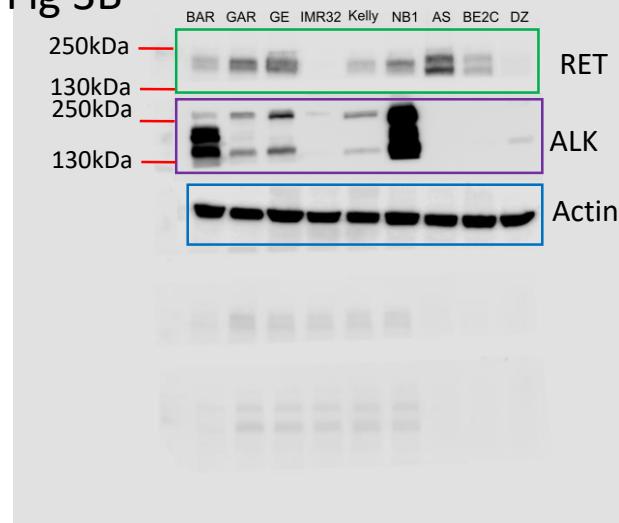


Fig 3C

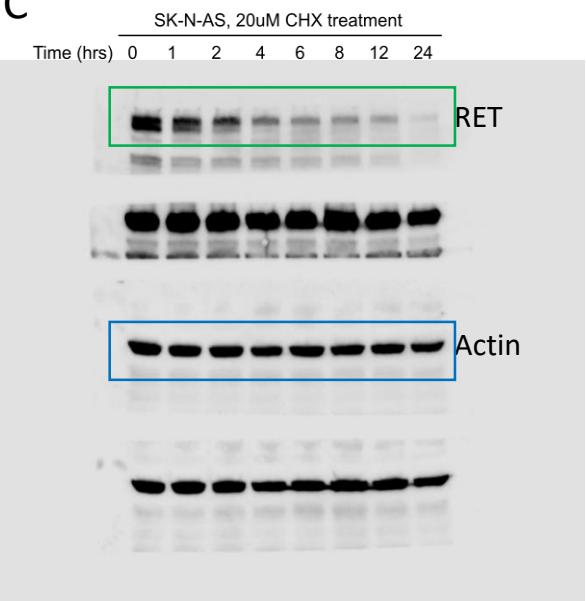


Fig 3D

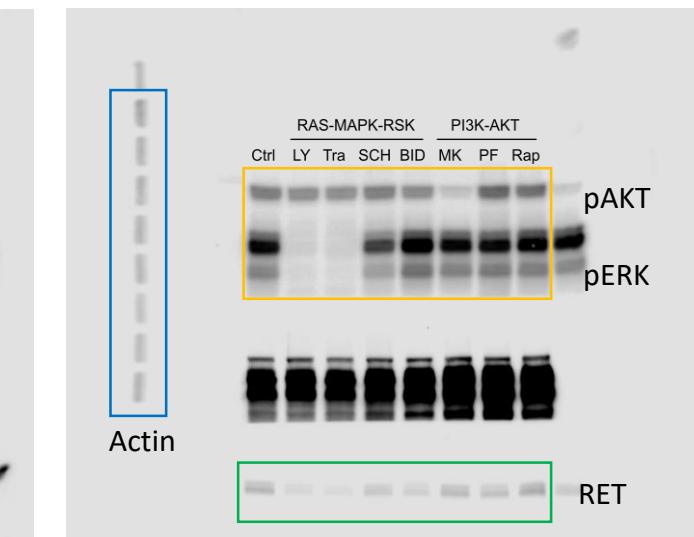
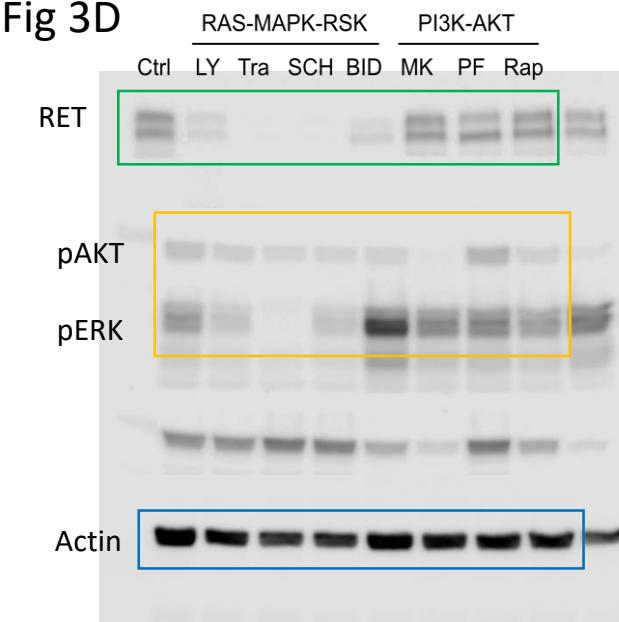


Fig. 3E-time course of treatment with tra and LY

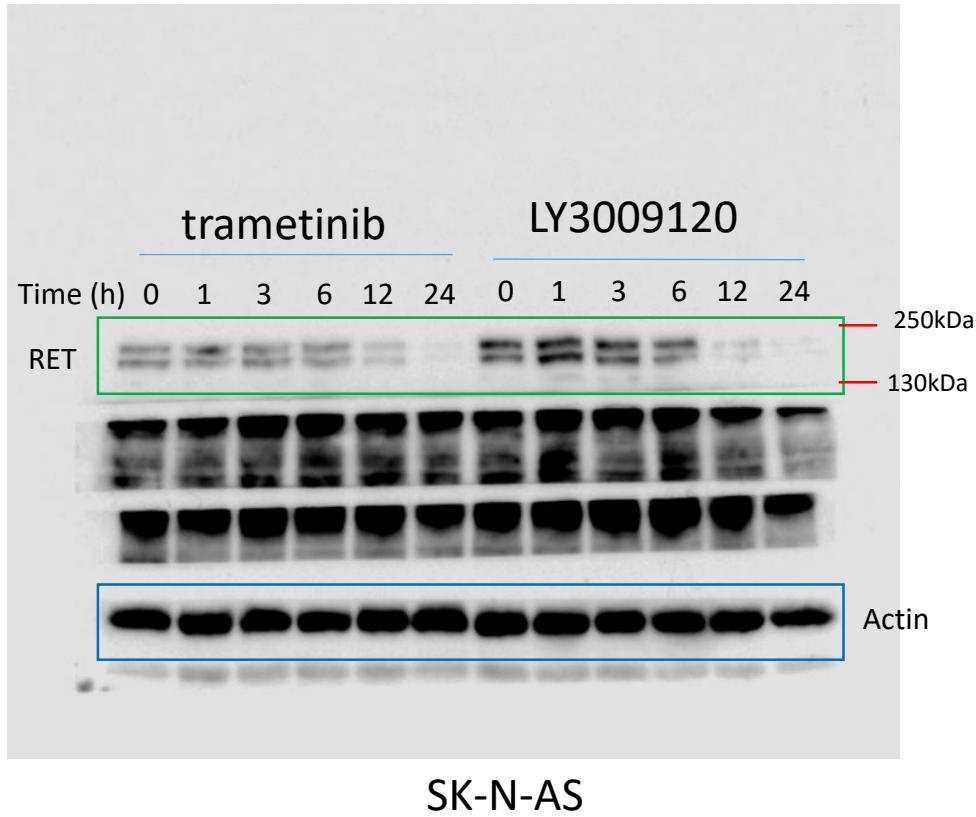


Fig. 4A-confirmation of RET expression in different clones

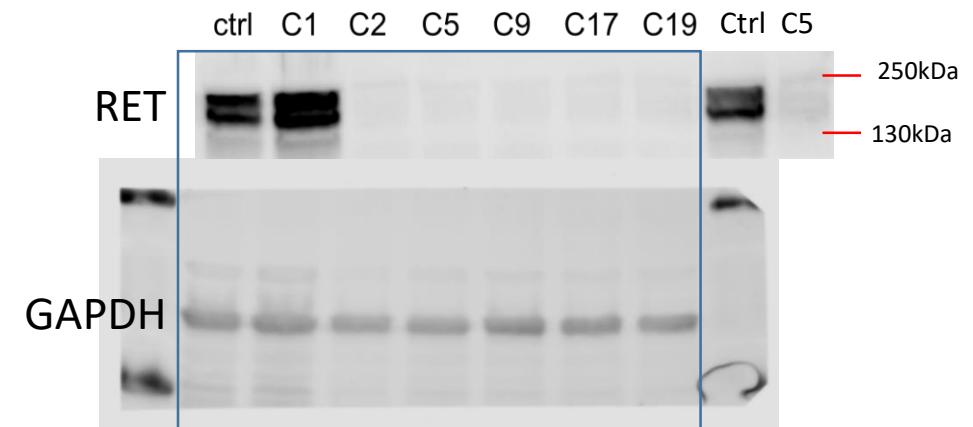


Fig. 7A

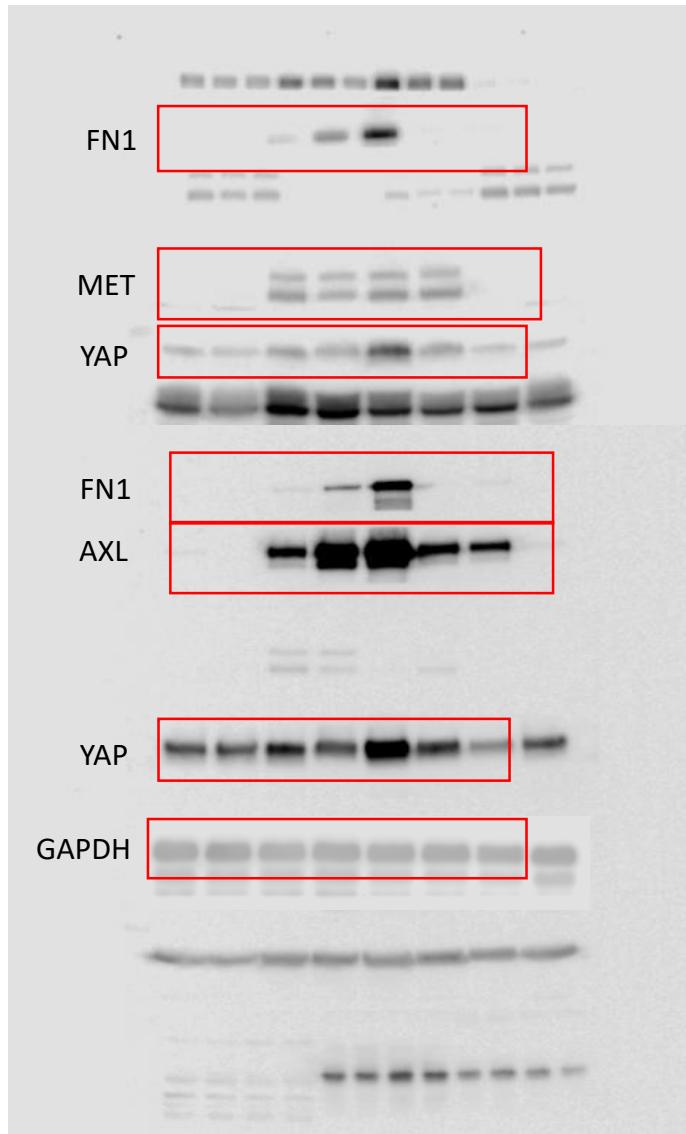


Fig. 7B

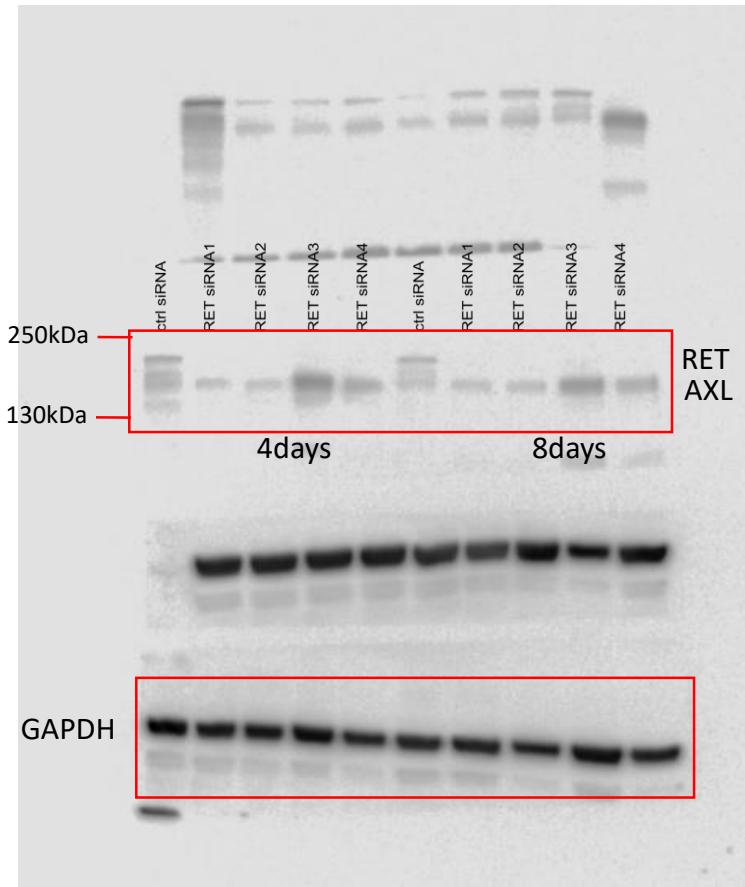


Fig. 7C

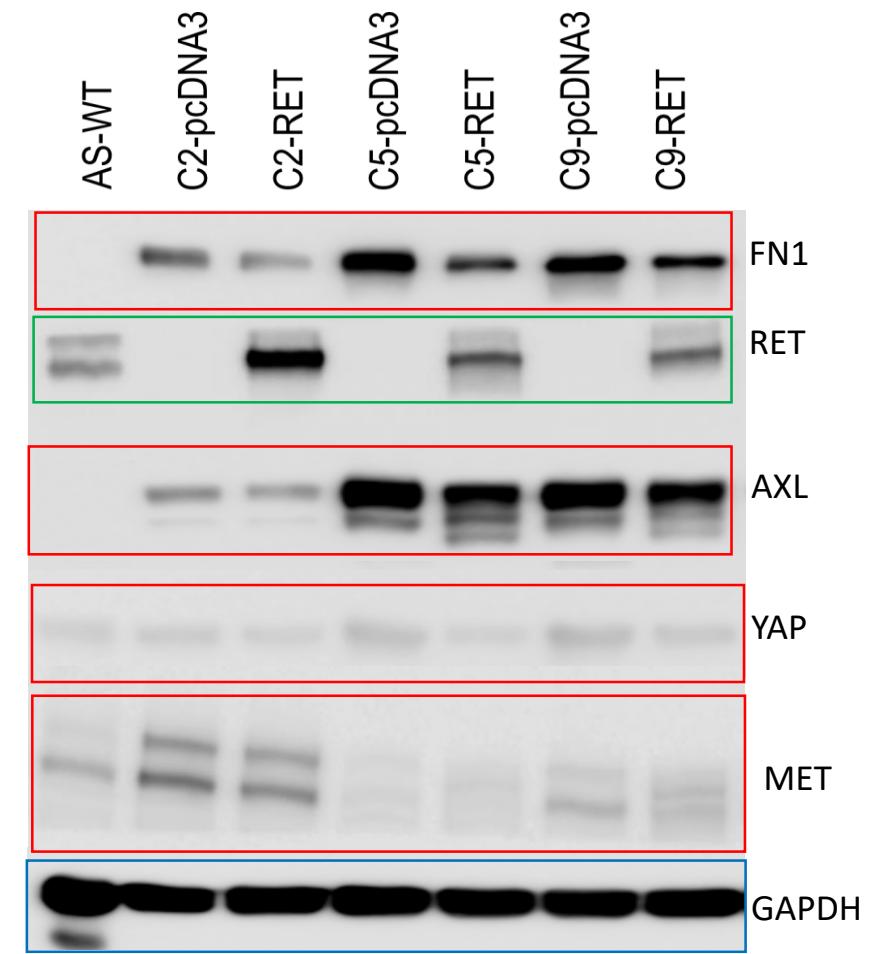


Fig. 9D

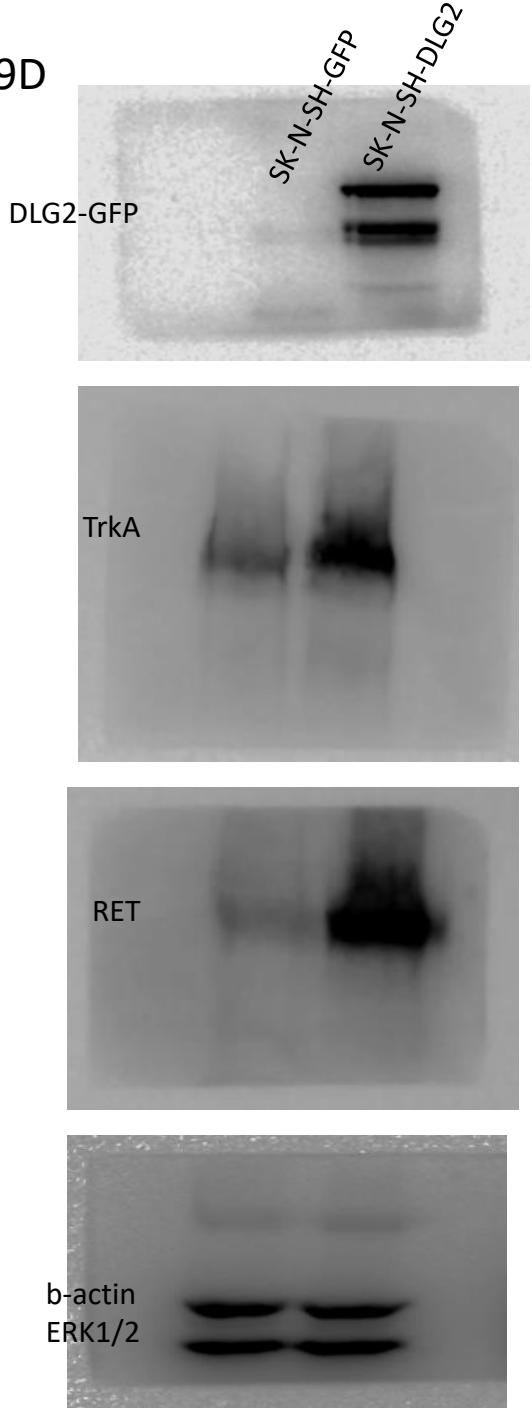


Fig. 9F

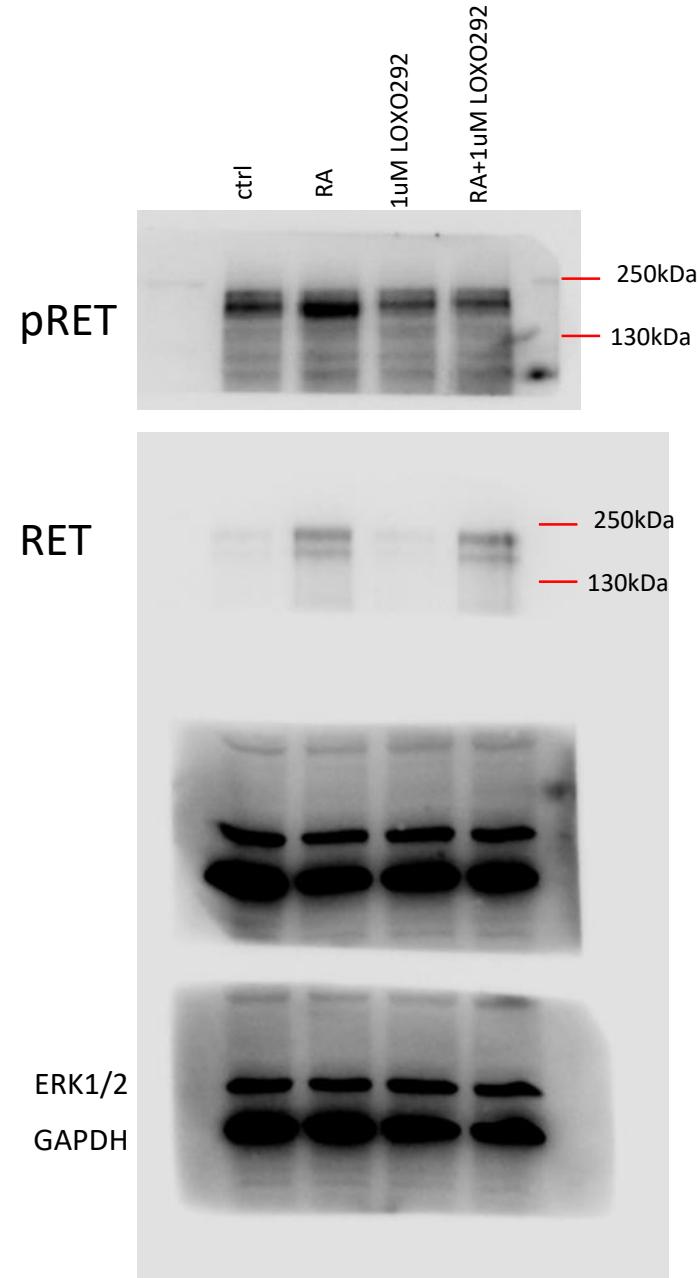
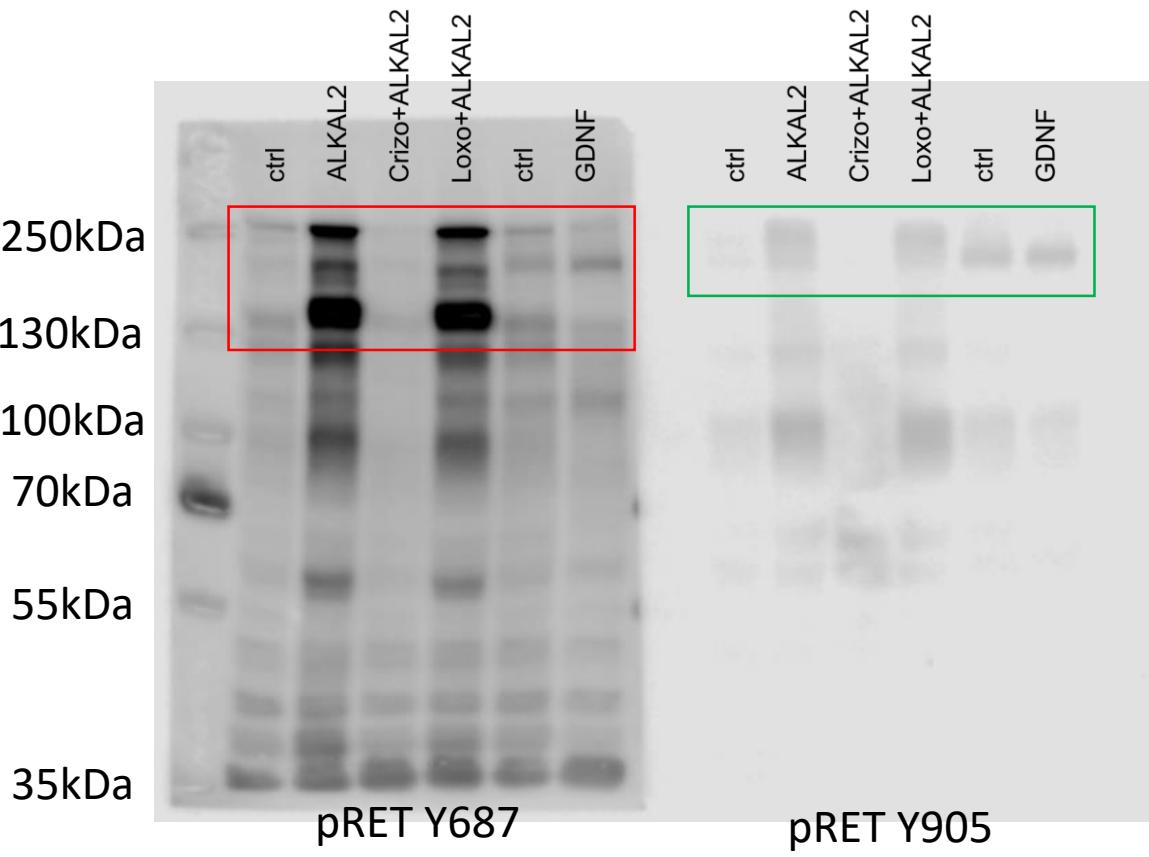


Fig S1-A: Cross-reaction of pRET antibodies to phosphorylated ALK



Due to hyperactivation of ALK upon stimulation with its ligand ALKAL2, pALK can also be detected with pRET antibodies. To avoid this cross-reaction, RET IP was performed to pull down total RET and then blot with pRET (see figure 1).

Fig. S1B: test ALK monoclonal antibody mAb53. The use of this antibody has not been published somewhere, so here to verify it.

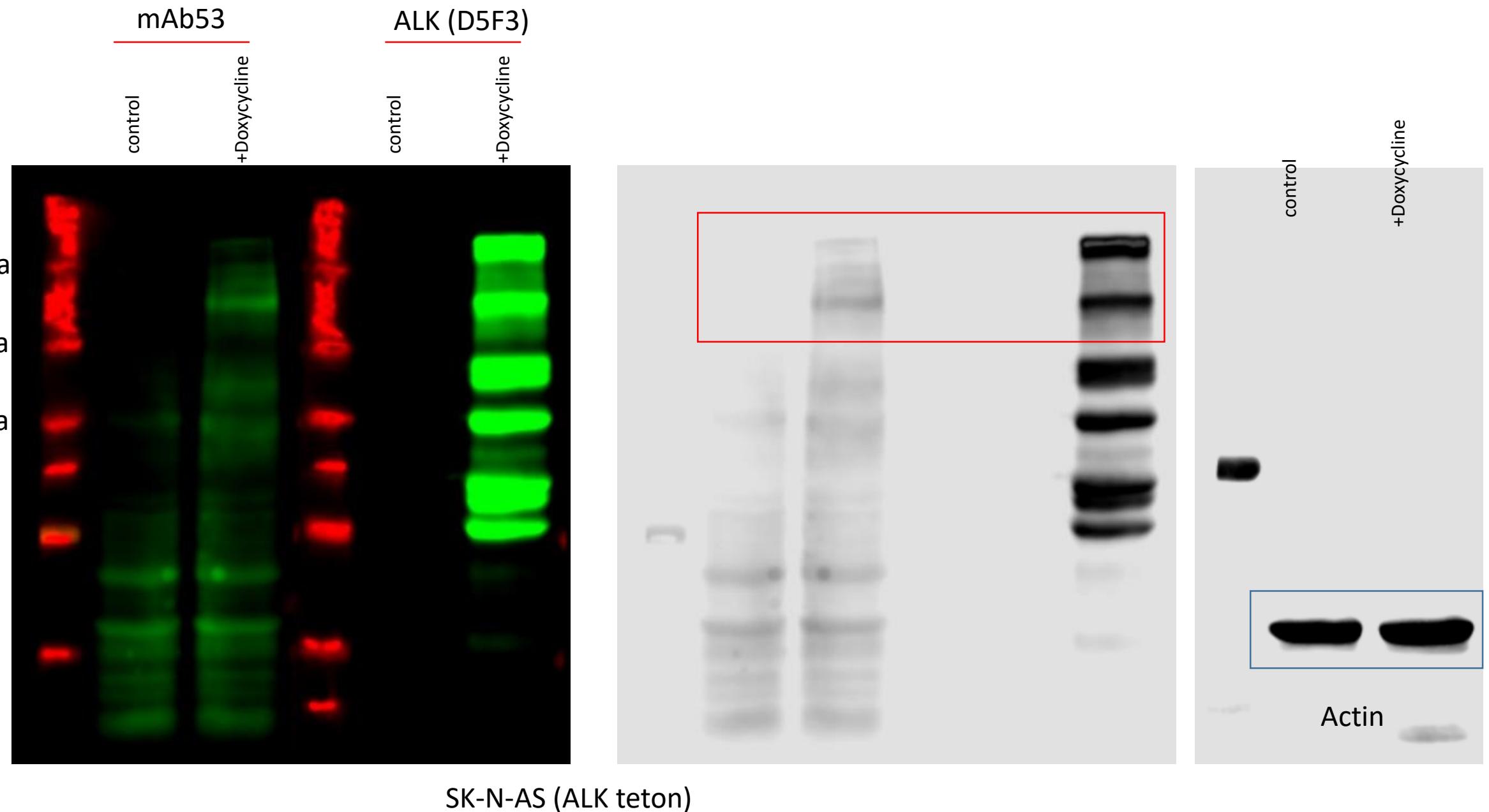


Fig S2. RET stimulation or inhibition in different ALK-driven NB cell lines.

