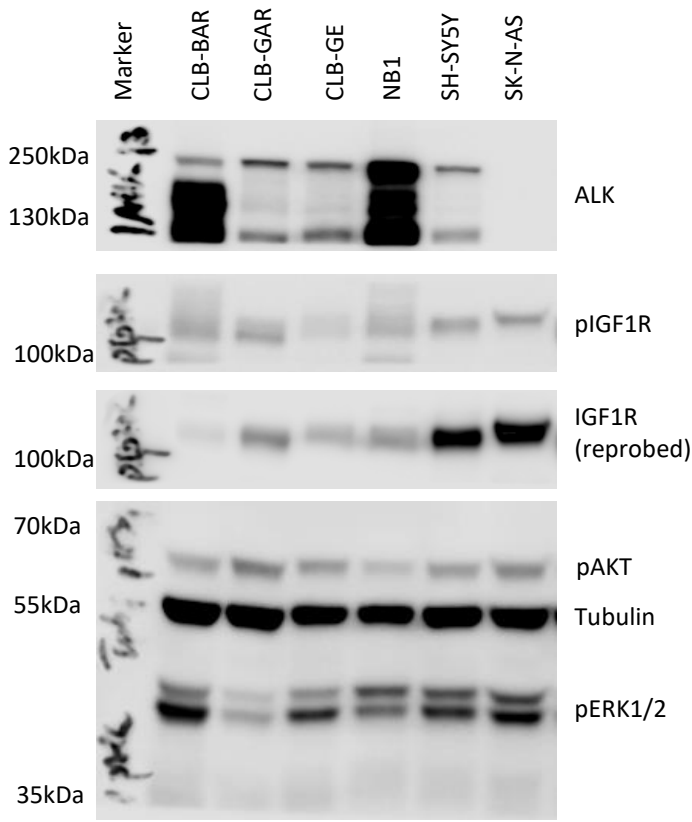


IGF1R-ALK-mutated NB

Raw data

Fig 1B. IGF1R acitivity is present in ALK-driven NB cells.



Revised figure

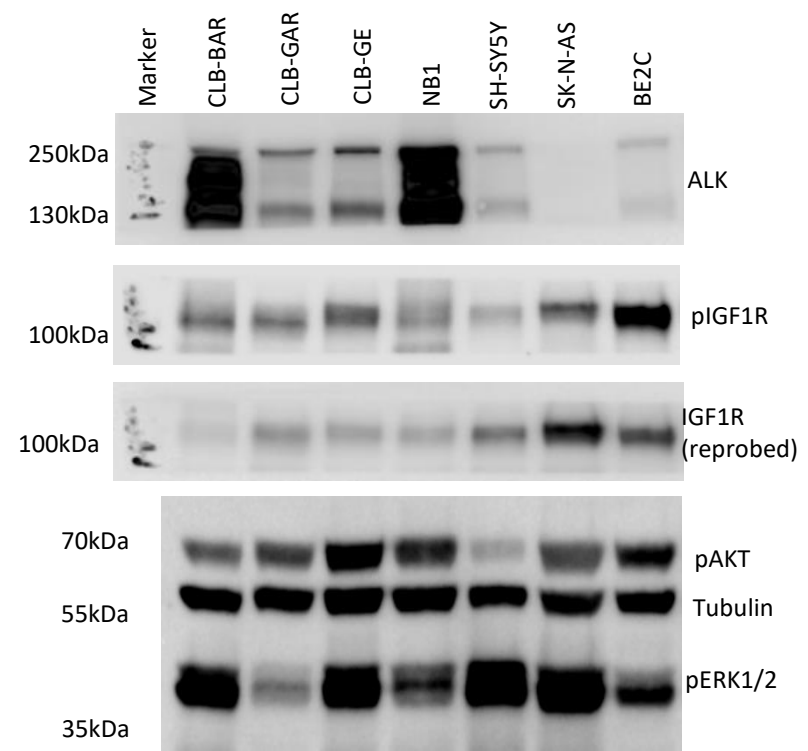
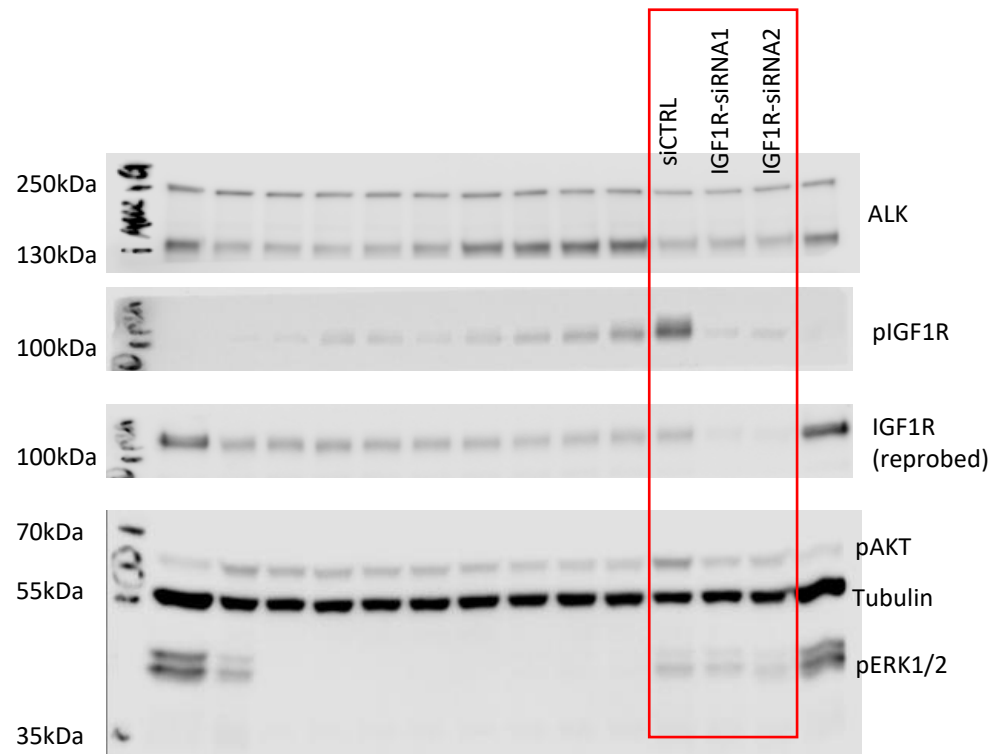
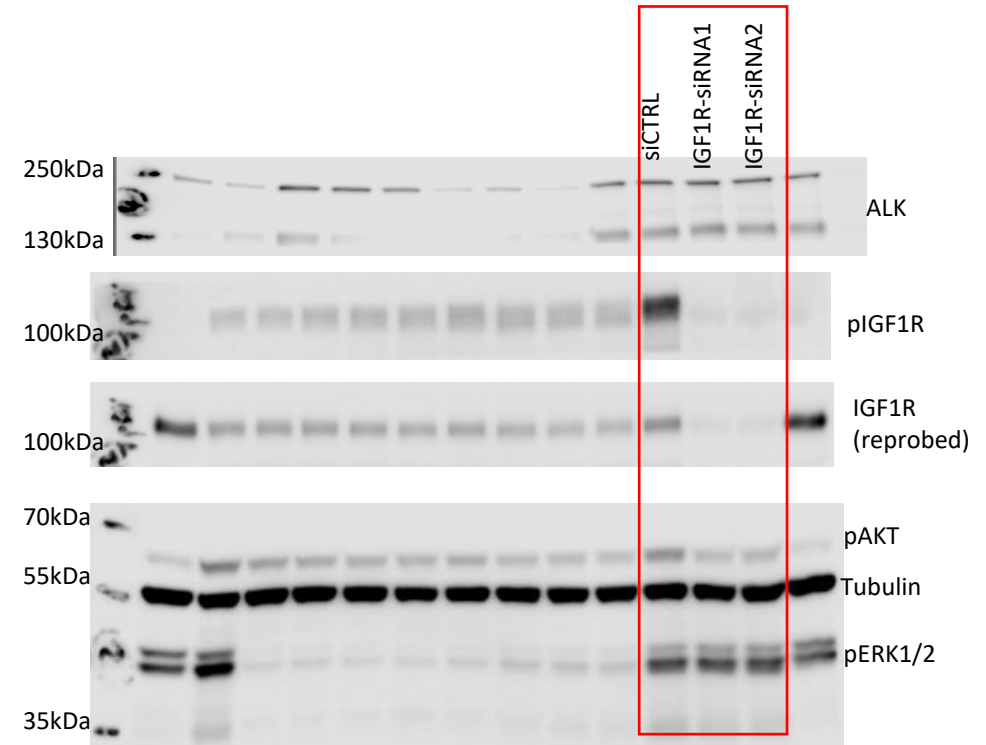


Fig 2F. Effects of knockdown of IGF1R in CLB-GAR and CLB-GE



CLB-GAR



CLB-GE

Fig 3A,B. BAR,GAR-GSK serial-inhibition

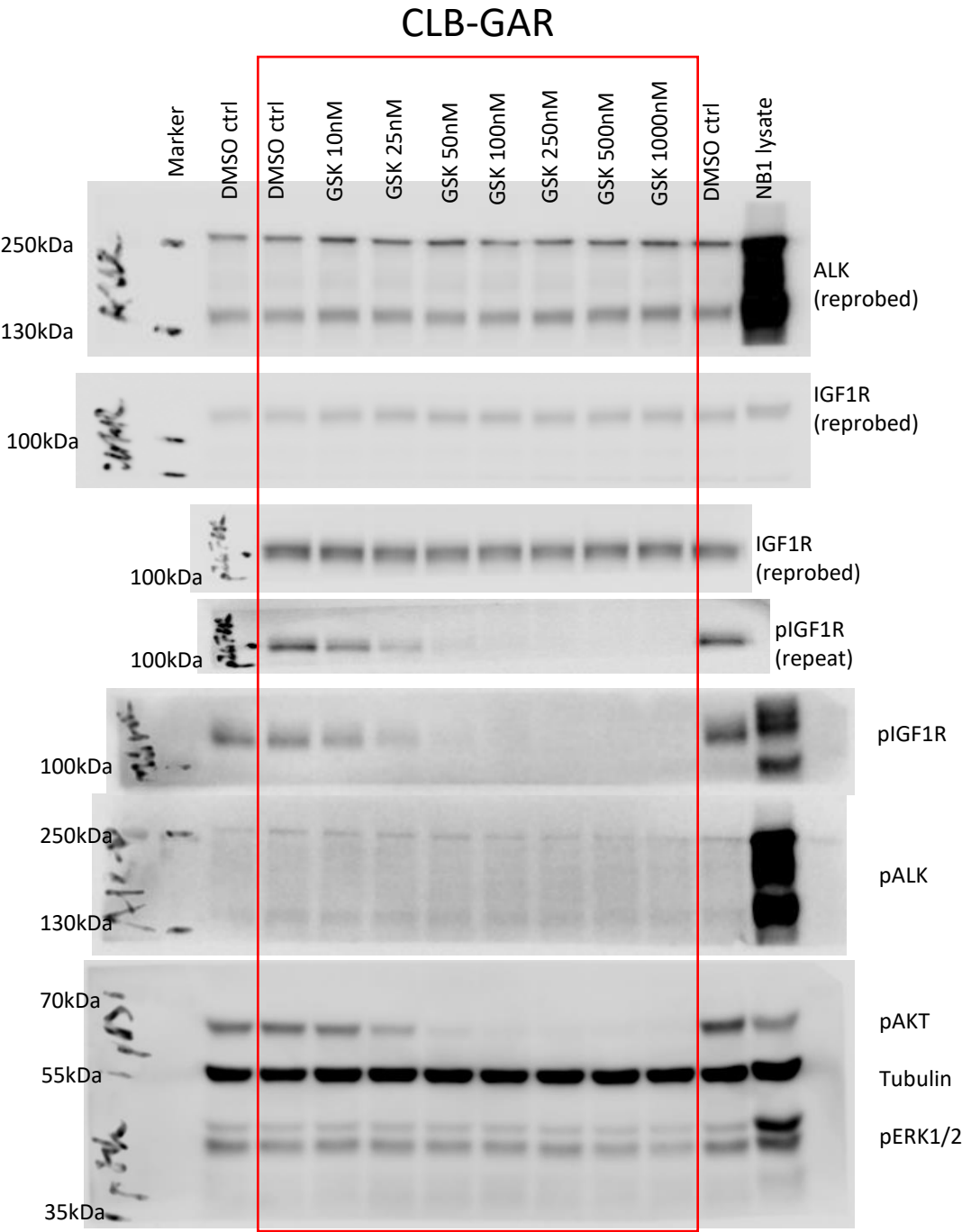
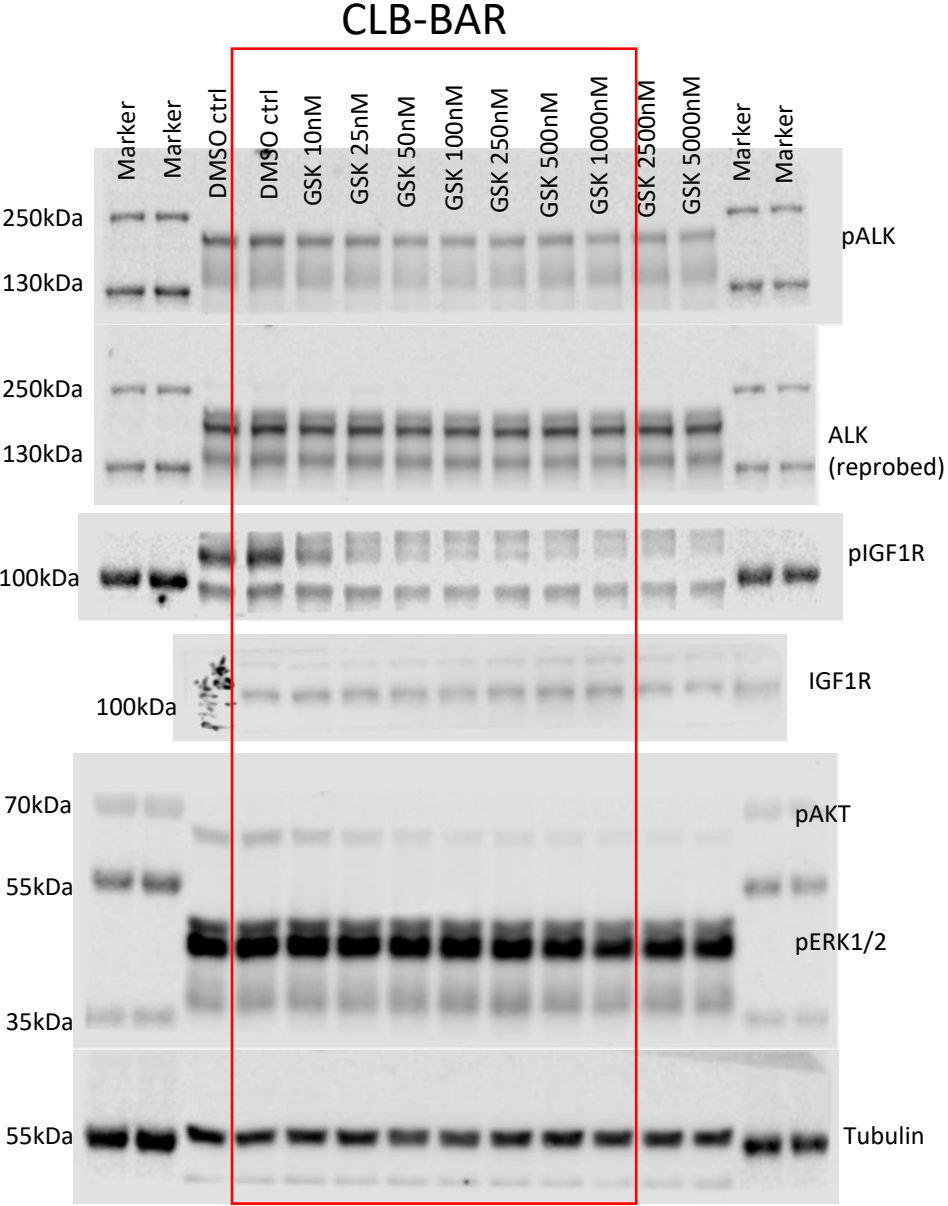


Fig 3,D. CLB-GE, NB1-GSK serial-Inhibition

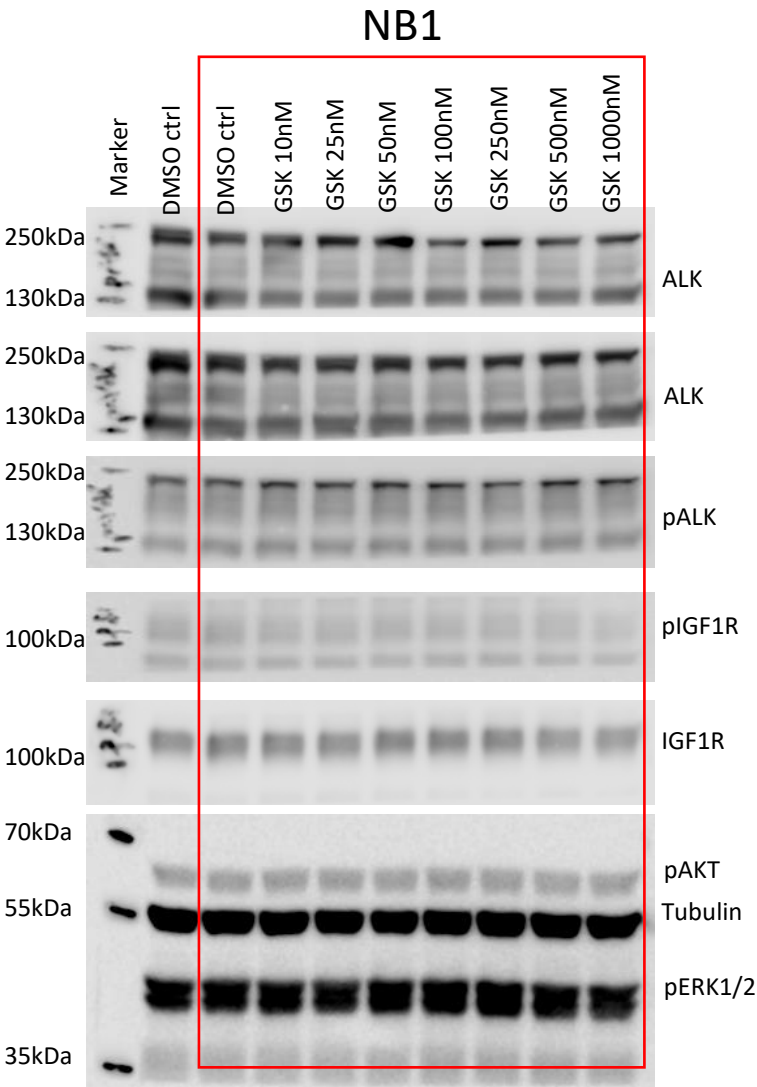
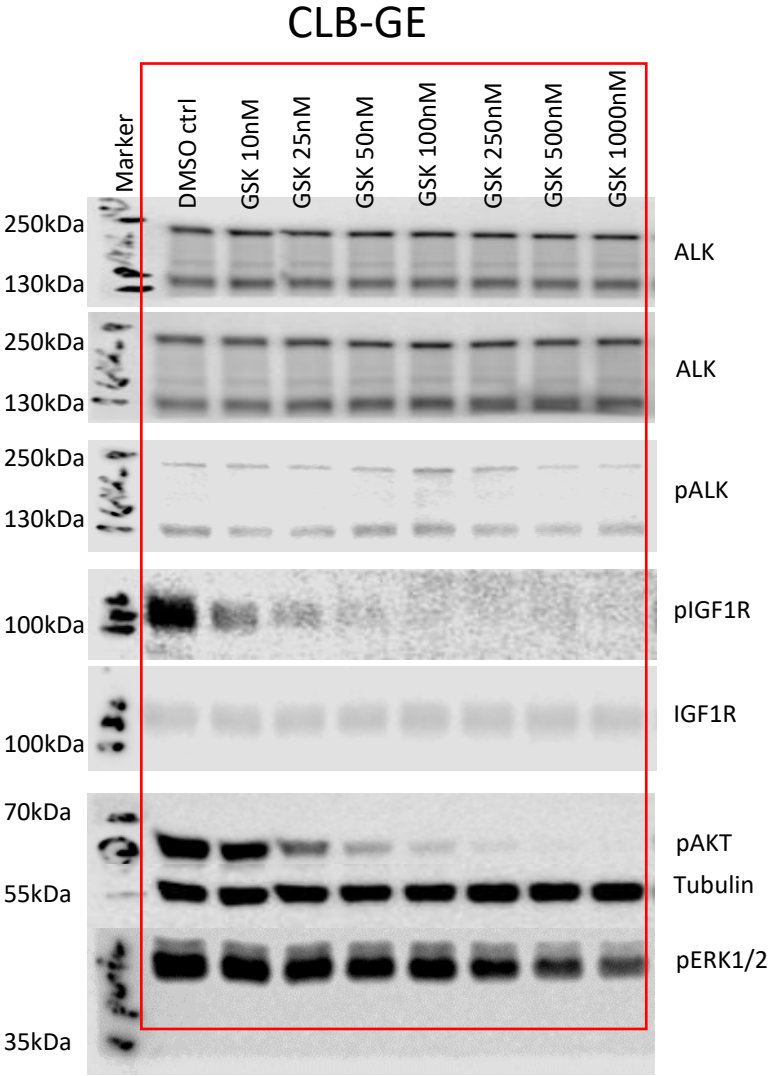


Fig 5. Both ALK and IGF1R activities contribute to downstream signaling (2h)

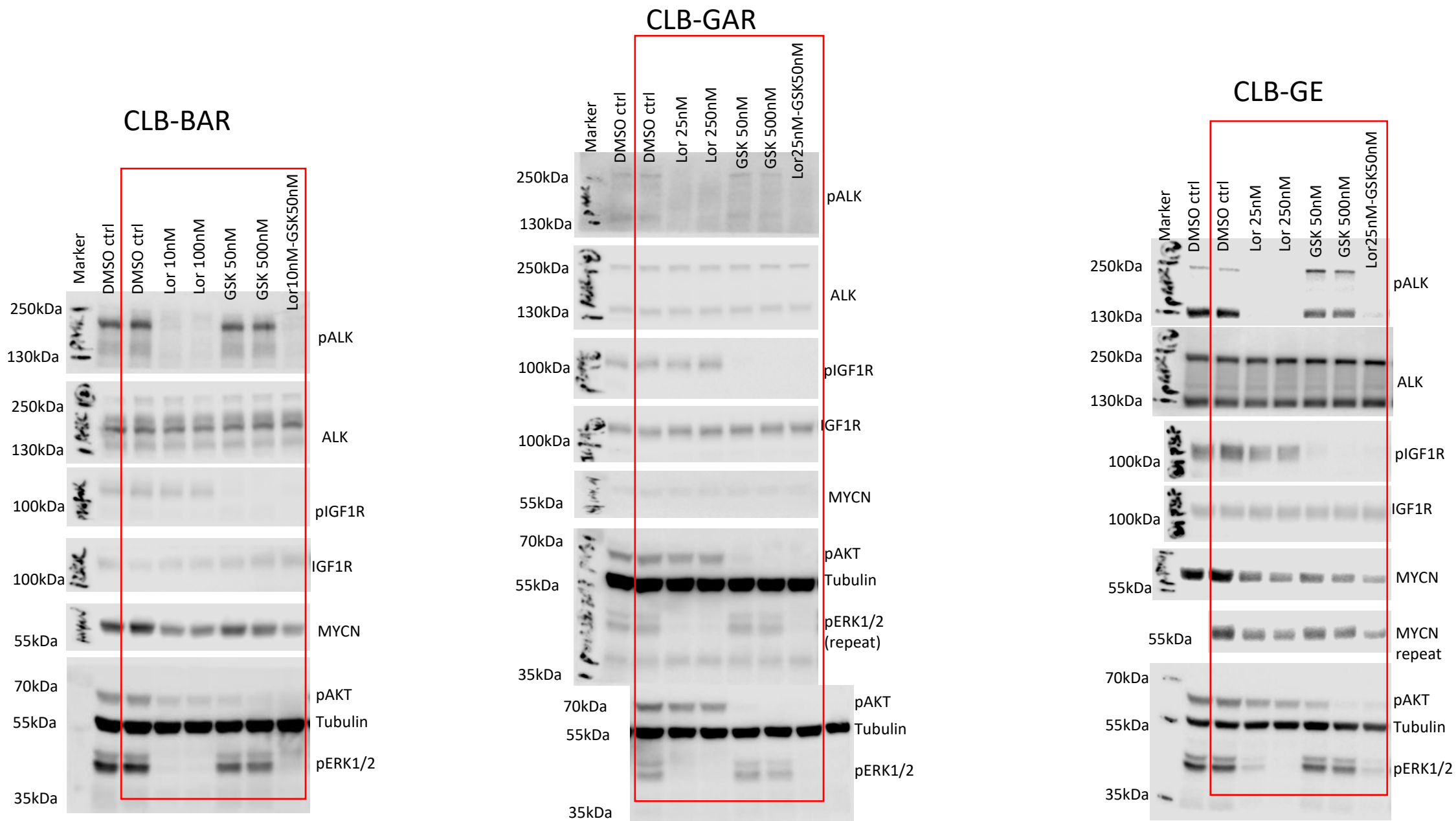


Fig 5. Both ALK and IGF1R activities contribute to downstream signaling (2h) (for revision)

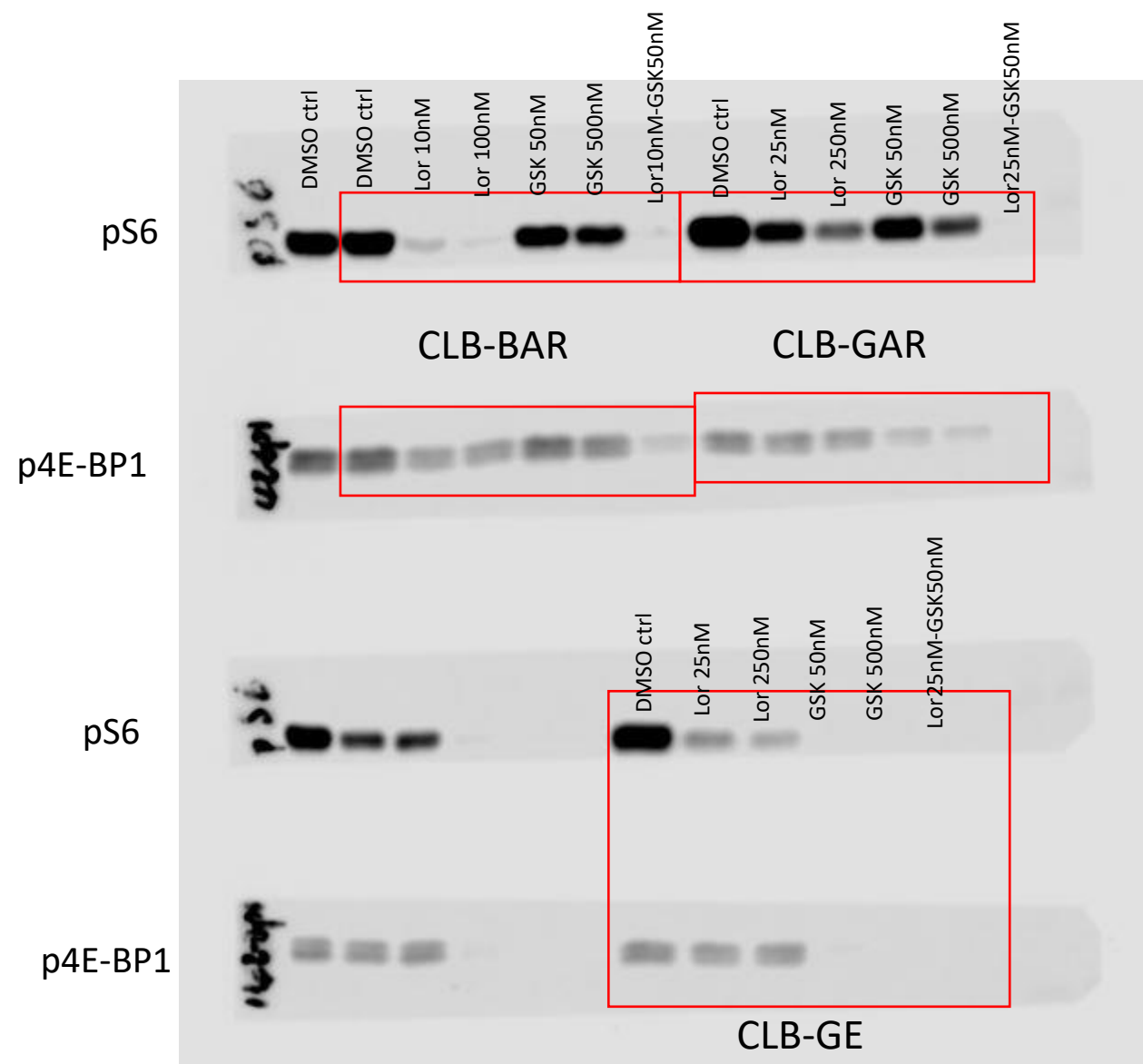


Fig 6. Differential preference for downstream adaptors

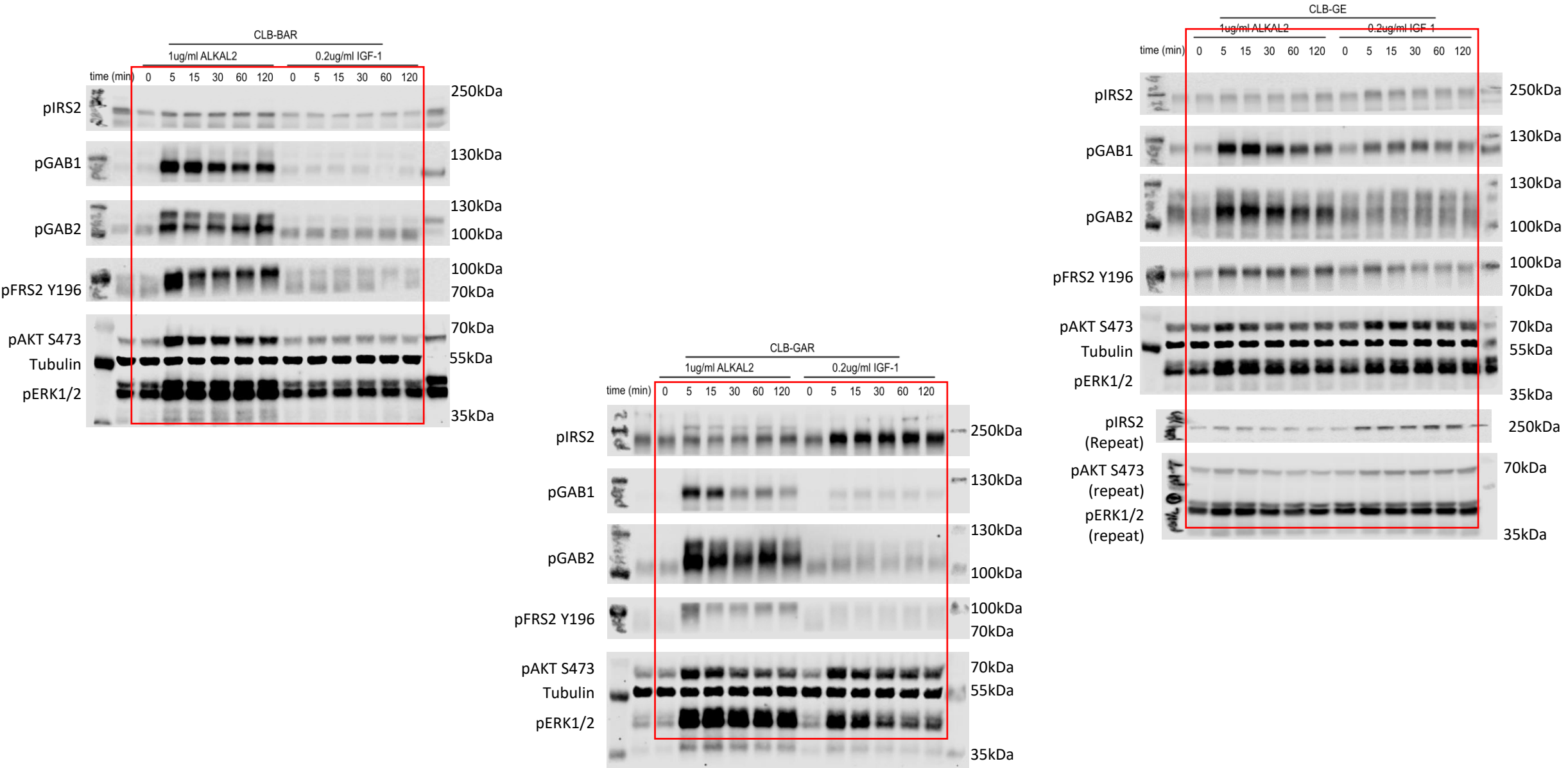
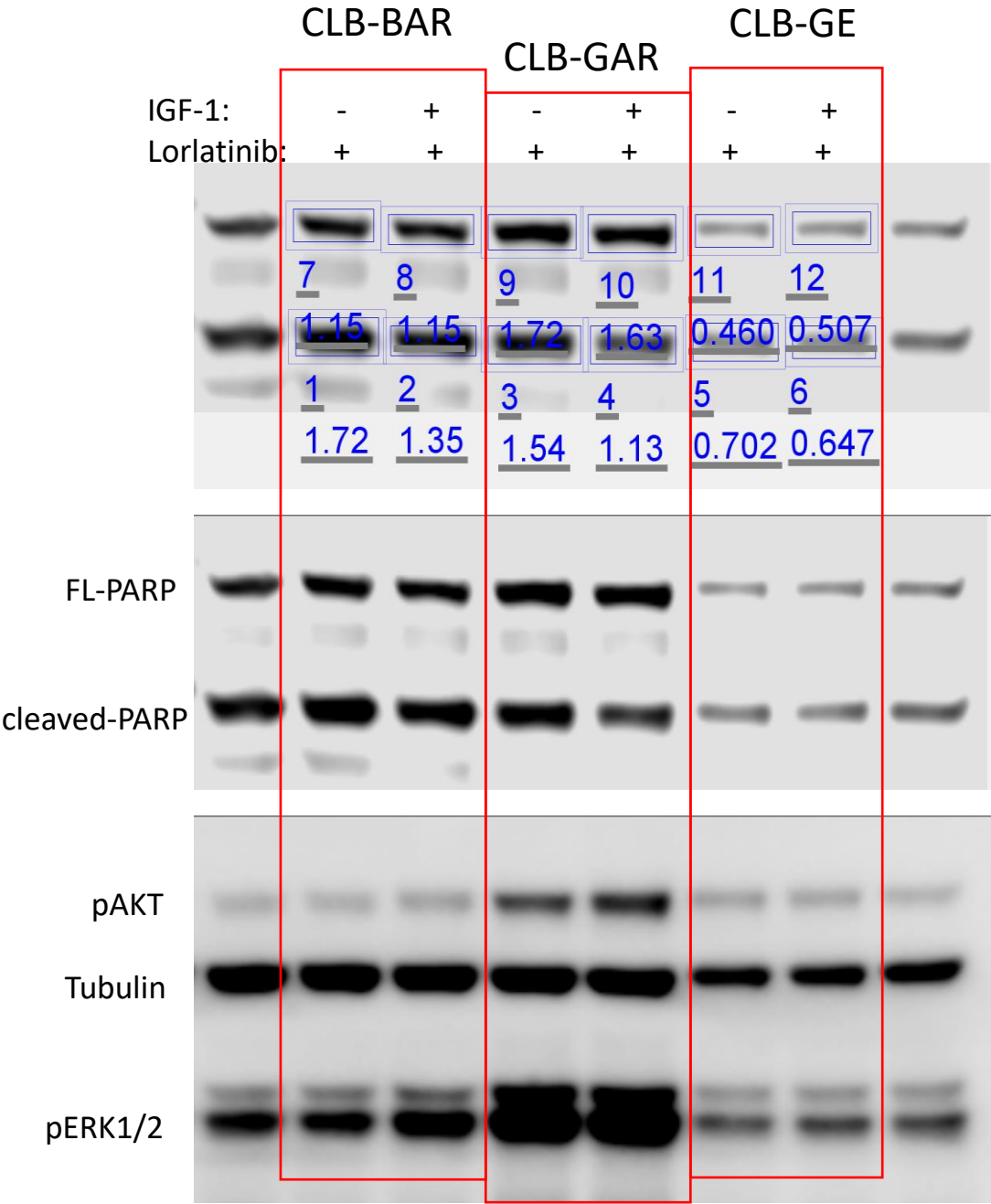


Fig S3B. Effect of IGF1R activity on cell apoptosis



Suppl. Fig4 Both ALK and IGF1R contribute to ALK-driven NBs

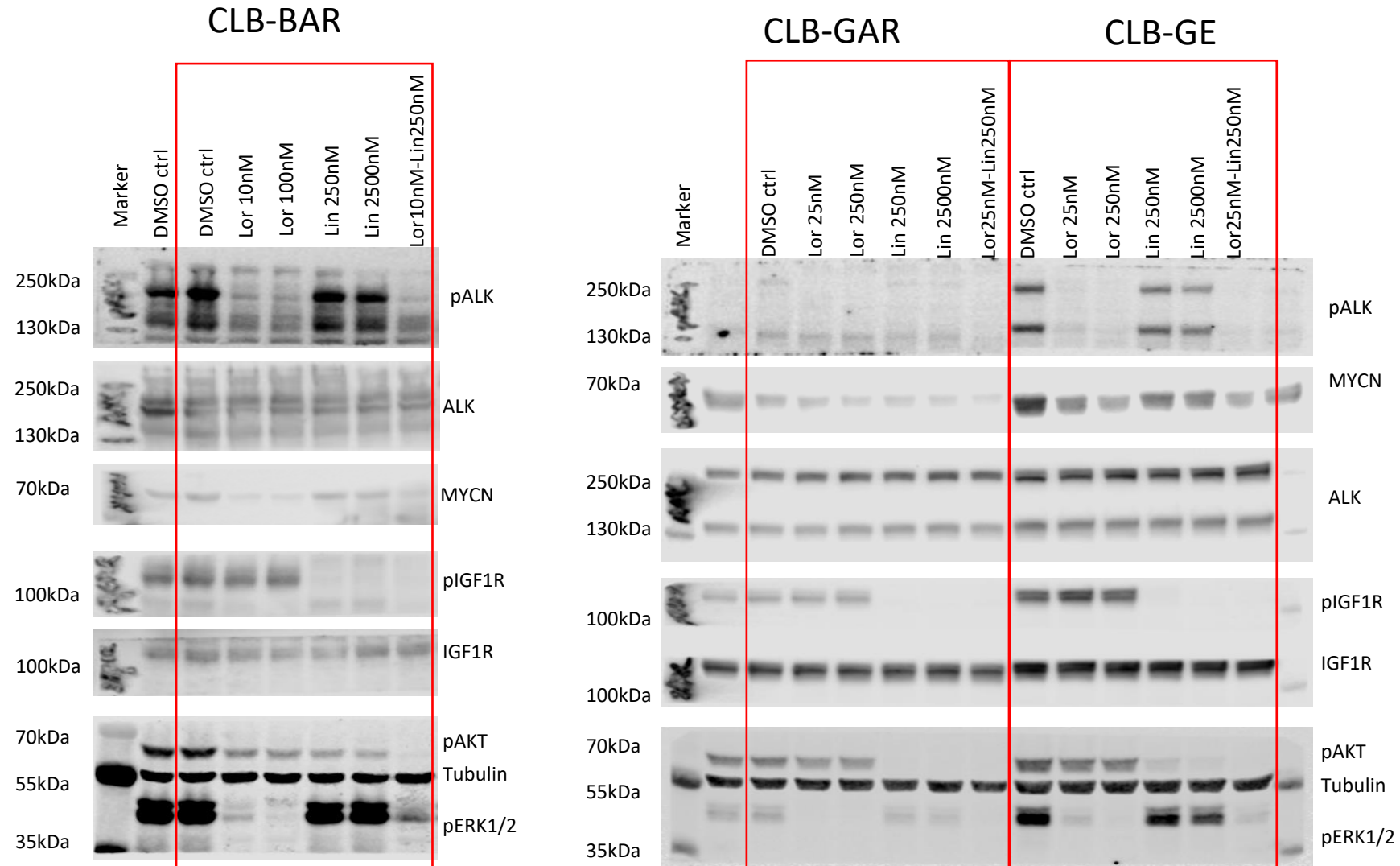


Figure S5. IRS2 recruits the PI3K p85 subunit upon stimulation with ALKAL2 or IGF-1.

