Identification of a novel oncogenic mutation of *FGFR4* in gastric cancer

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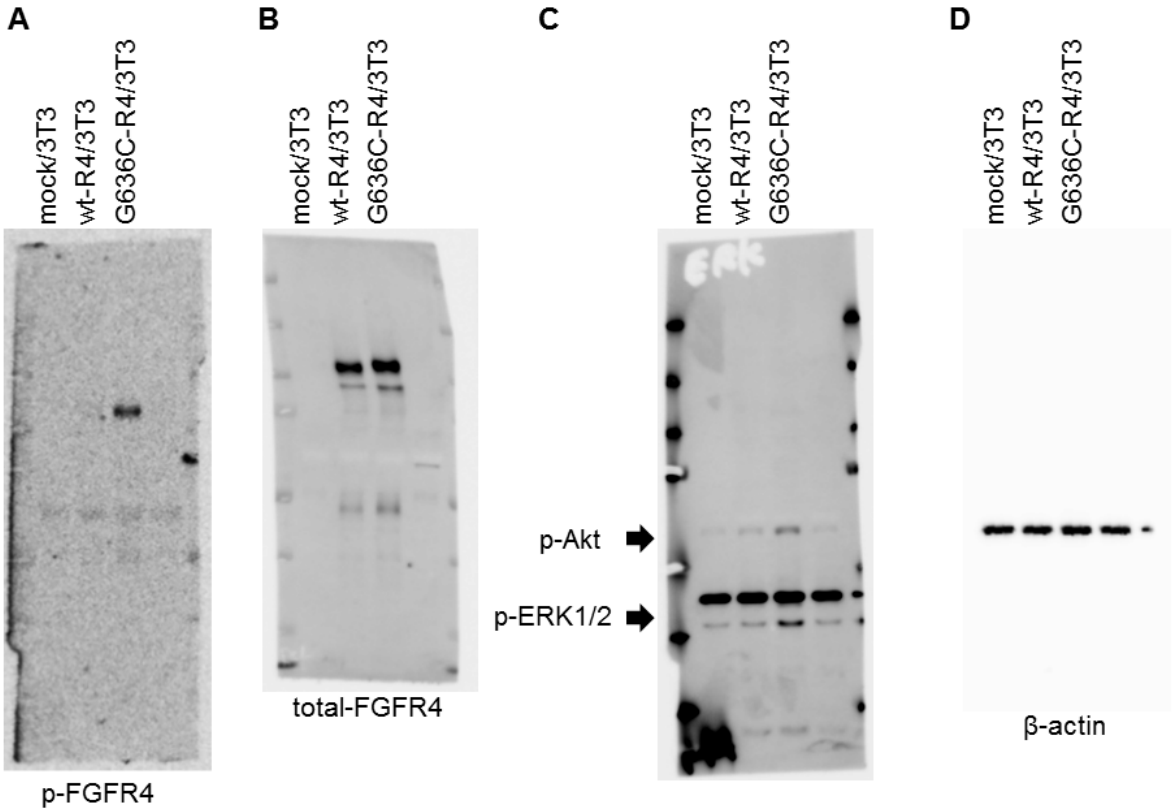
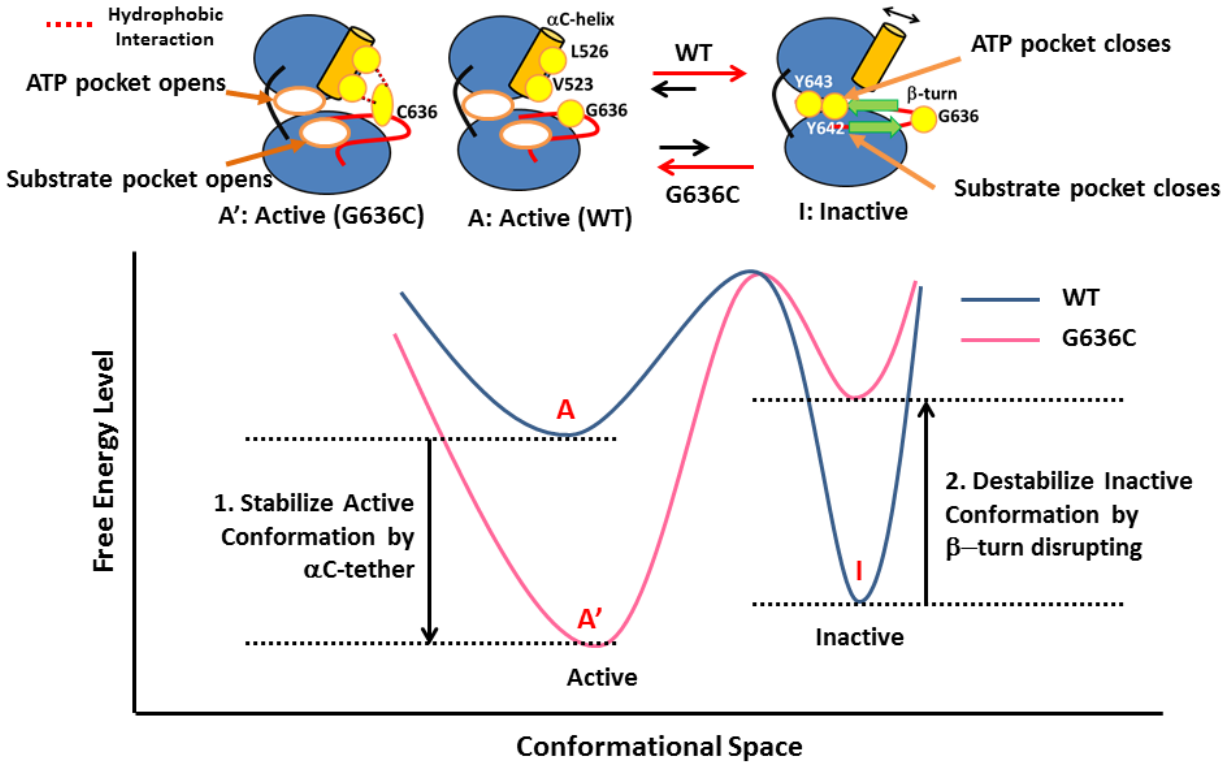
Supplementary Information

Fig. S1 Full gel images of Fig. 3A



Expression and phosphorylation of FGFR4 and downstream signalling molecules A) phospho-FGFR4, B) FGFR4, C) phospho-ERK1/2, phospho-AKT and D) β-actin

Fig. S2. Effects of the FGFR4 G636C mutation on the equilibrium between active and inactive conformations.



The kinase domain of FGFR4 is in equilibrium between active and inactive conformations. The probability of the formation of these conformations depends on the free energy level. The lower the free energy of a conformation, the higher its probability. The G636C mutation shifts the equilibrium to the active conformation through the following effects: (1) stabilization and lowering the free energy level of the active conformation through hydrophobic interactions among C636, V523, and L526 (αC-tether), (2) destabilization and increasing the free energy of the inactive conformation by distrupting the β-turn.