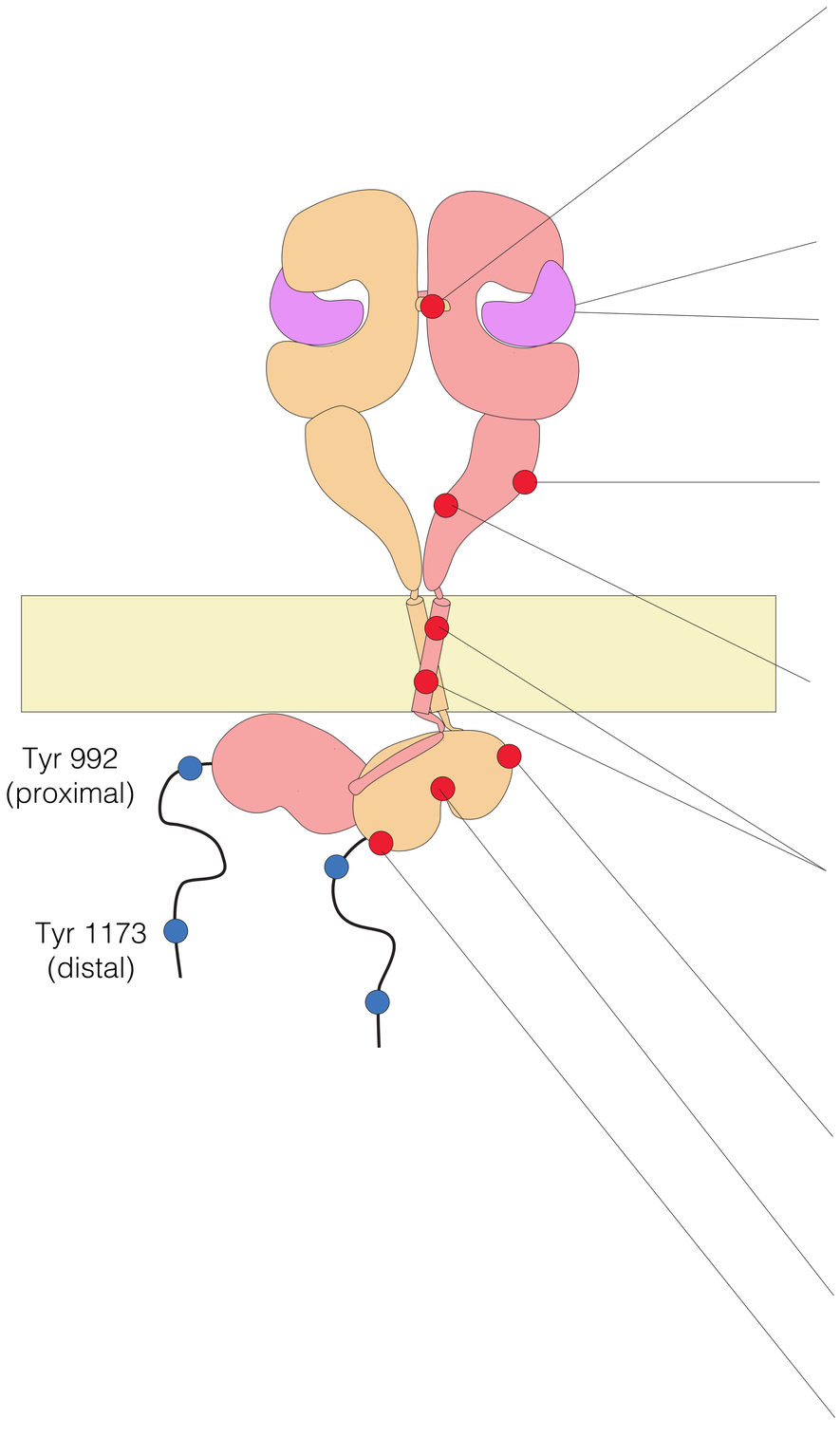
Figure 11,
Supplemental Figure 1



Mutation or Variation	Effect on Activity	Effect on Multimerization
deletion of dimerization arm: Δ-arm	strong reduction of autophosphorylation in Tyr 992 and Tyr 1173	increases EGF-independent dimerization but has little effect on EGF-induced multimerization modest effect on EGF-induced dimerization
replacement of EGF by TGF-α		no reduction in EGF-induced multimerization
EGF concentration increased from 15 nM to 2 μM		no reduction in EGF-induced multimerization
mutations in the proposed Domain IV multimerization interface: IIIV/KKRE VEN/ERR TN/RR	reduction in Tyr 992 and Tyr 1173 phosphorylation strong reduction of PI3K phosphorylation no effect on ERK phosphorylation	decreases EGF-dependent multimerization no reduction in EGF-induced dimerization
mutation of the autoinhibitory tether on Domain IV: Δ-tether		increases EGF-independent dimerization
mutations in the transmembrane helix (small residues in the N- and C-terminal interfaces replaced by isoleucine)	modest effect on Tyr 1173 stronger effect on Tyr 992 strong reduction of PI3K phosphorylation no effect on ERK phosphorylation	strong reduction of EGF-dependent multimerization no reduction in EGF-induced dimerization
receiver-impaired (I682Q)	tail can only be phosphorylated within an asymmetric dimer	reduces EGF-dependent multimerization and EGF-independent dimerization when combined with V924R
kinase-dead (D813N)	tail can be phosphorylated only within an asymmetric dimer	no effect on EGF-dependent multimerization
activator-impaired (V924R)		reduces EGF-dependent multimerization and EGF-independent dimerization when combined with I682Q