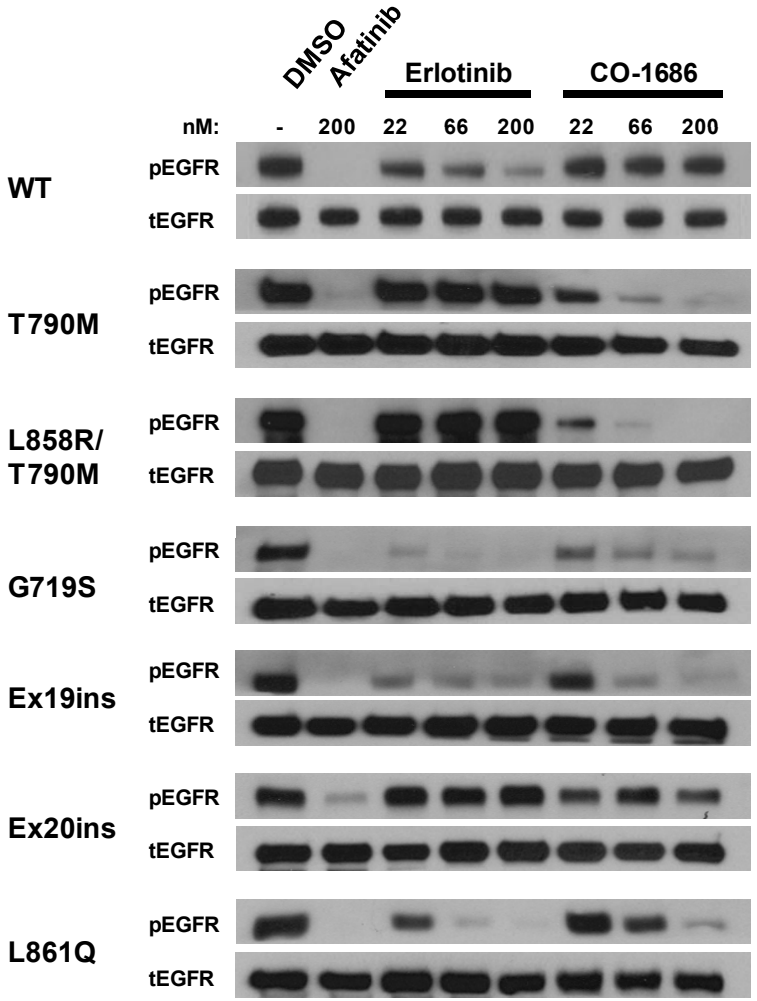
Supplemental Figure 3



SI figure 3. Activity of CO-1686 against rare lung-cancer associated EGFR mutants. 293H cells were transiently transfected with cDNAs encoding various mutant EGFRs. Following 6 hour treatment with DMSO, afatinib, or increasing doses of erlotinib or CO-1686 at the indicated concentrations, cells were lysed. Immunoblotting was performed using corresponding lysates with antibodies against phospho- and total EGFR. As expected, CO-1686 was more active against EGFR T790M and L858R + T790M than erlotinib. CO-1686 was also active against G719S, an exon 19 insertion mutant (ex19ins: I744-K745insKIPVAI), and L861Q, but not against an exon 20 insertion (ex20ins: (H773-V774HVdup)). Afatinib was used at a supratherapeutic dose (200 nM) as a positive control for EGFR inhibition.