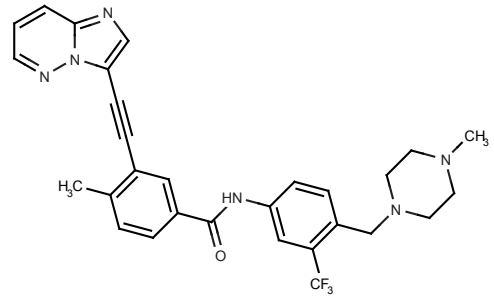
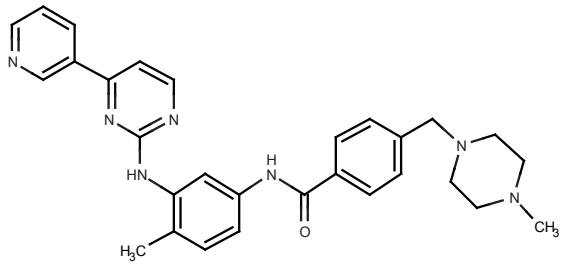
Supplementary figures 1-6

Figure S1. Chemical structures of imatinib, sunitinib, regorafenib and ponatinib

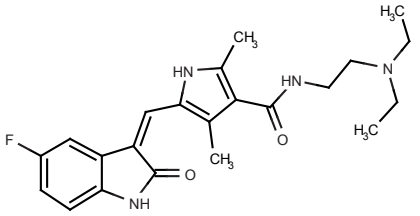
Ponatinib



Imatinib



Sunitinib



Regorafenib

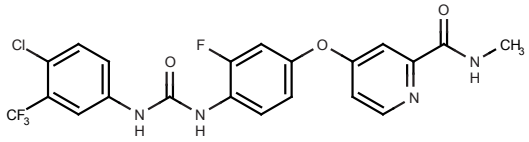


Figure S2. Expression and activation of KIT in engineered Ba/F3 cells

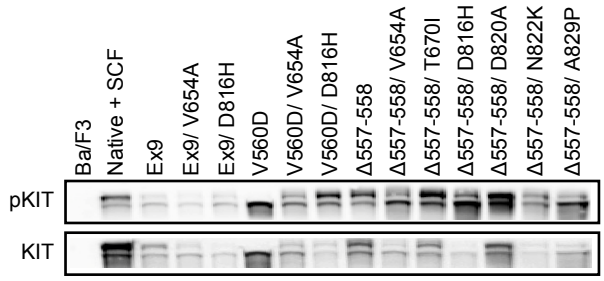


Figure S3. Ponatinib inhibits the phosphorylation of exon 11primary activating, and secondary resistant mutant forms of KIT

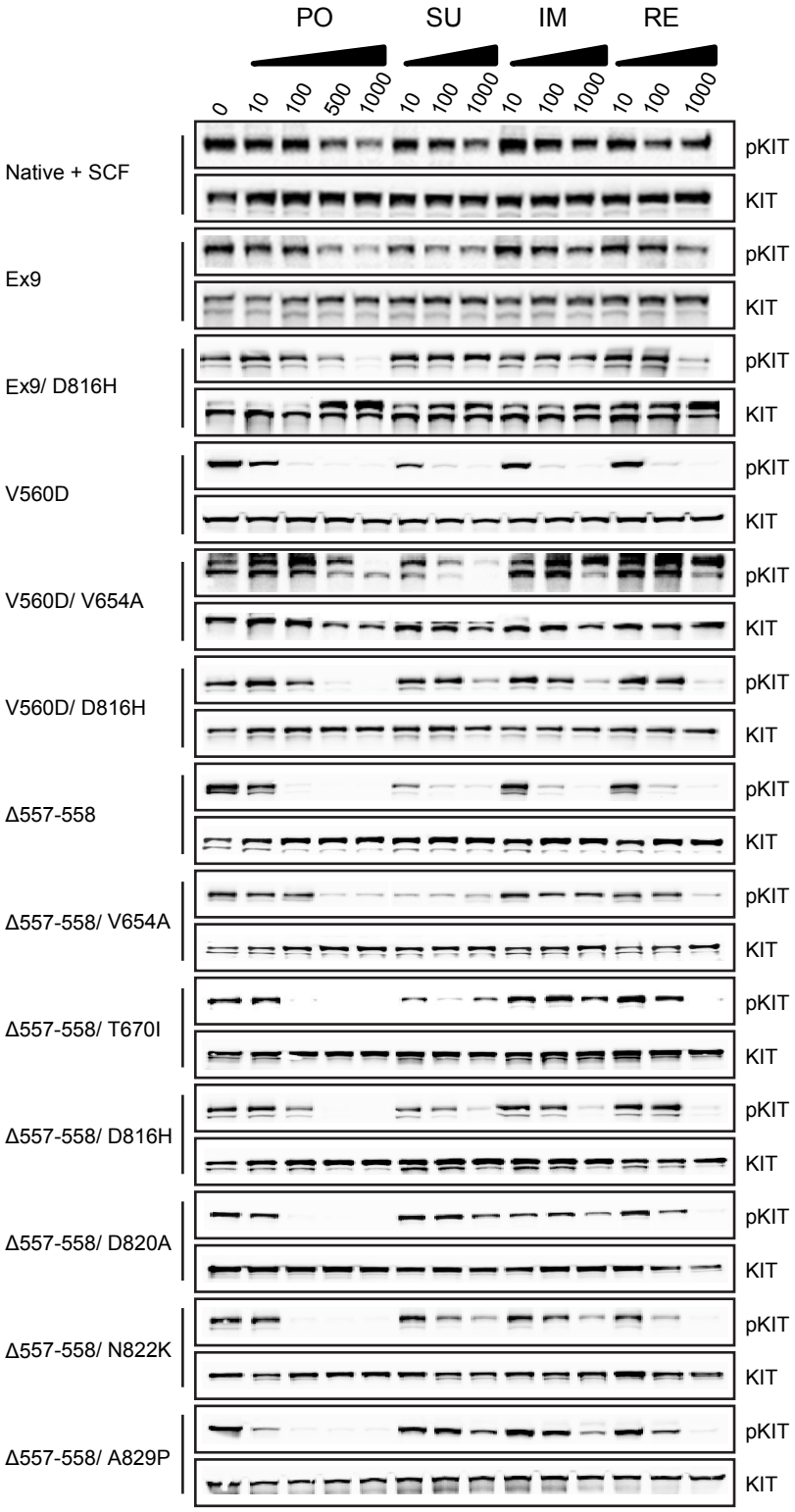
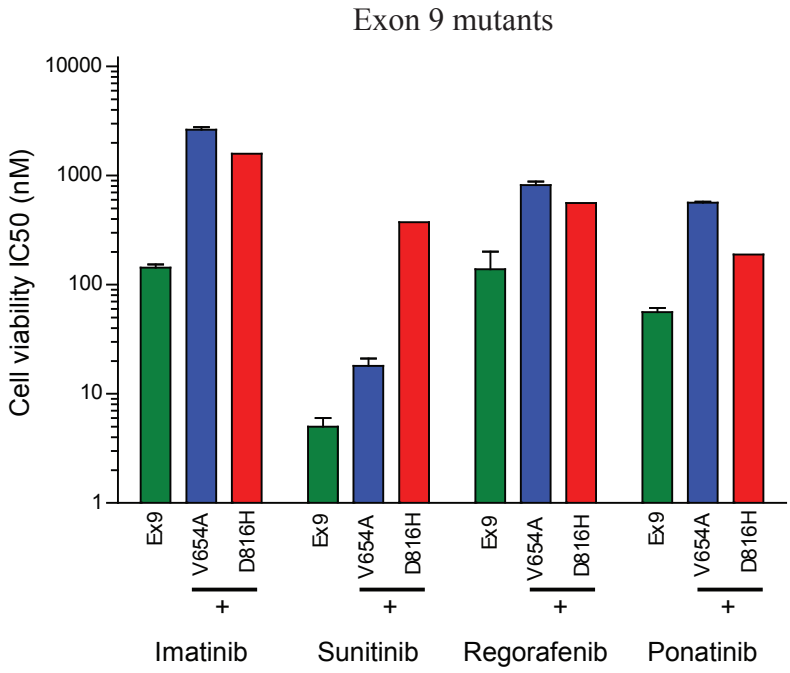


Figure S4. Secondary mutants reduce ponatinib potency in Ex9 ins and V560D cell lines

A



B

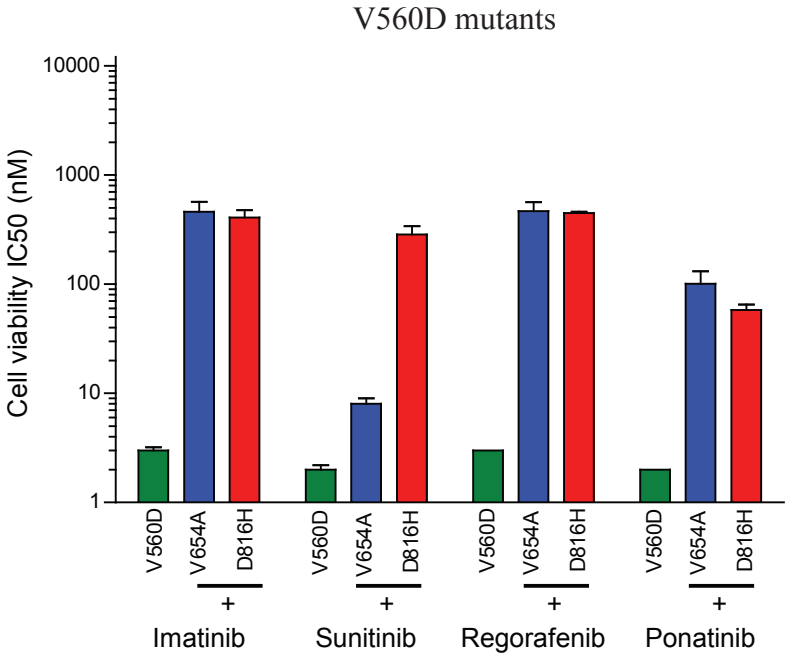


Figure S5. Illustration of the optimal fit of ponatinib to KIT

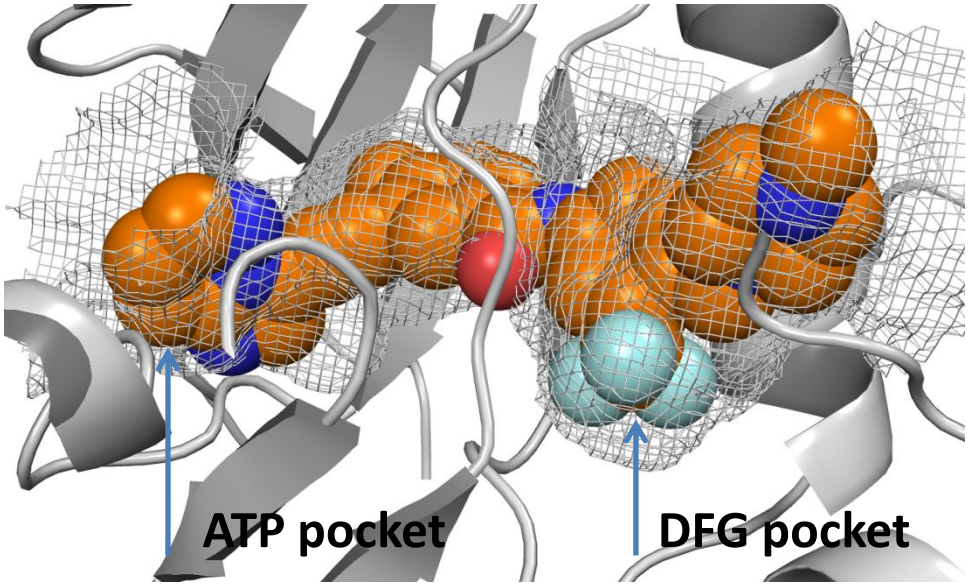
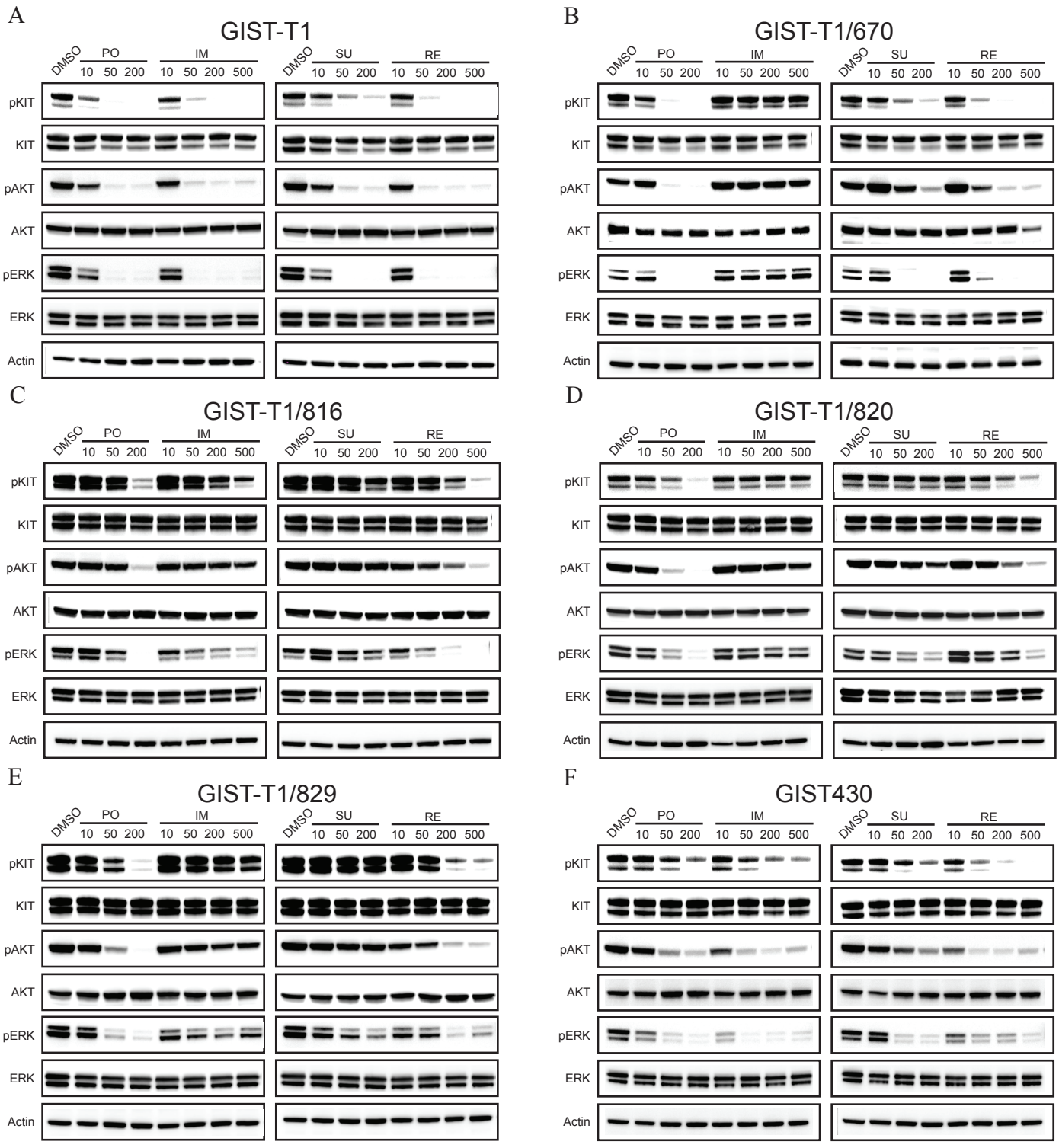
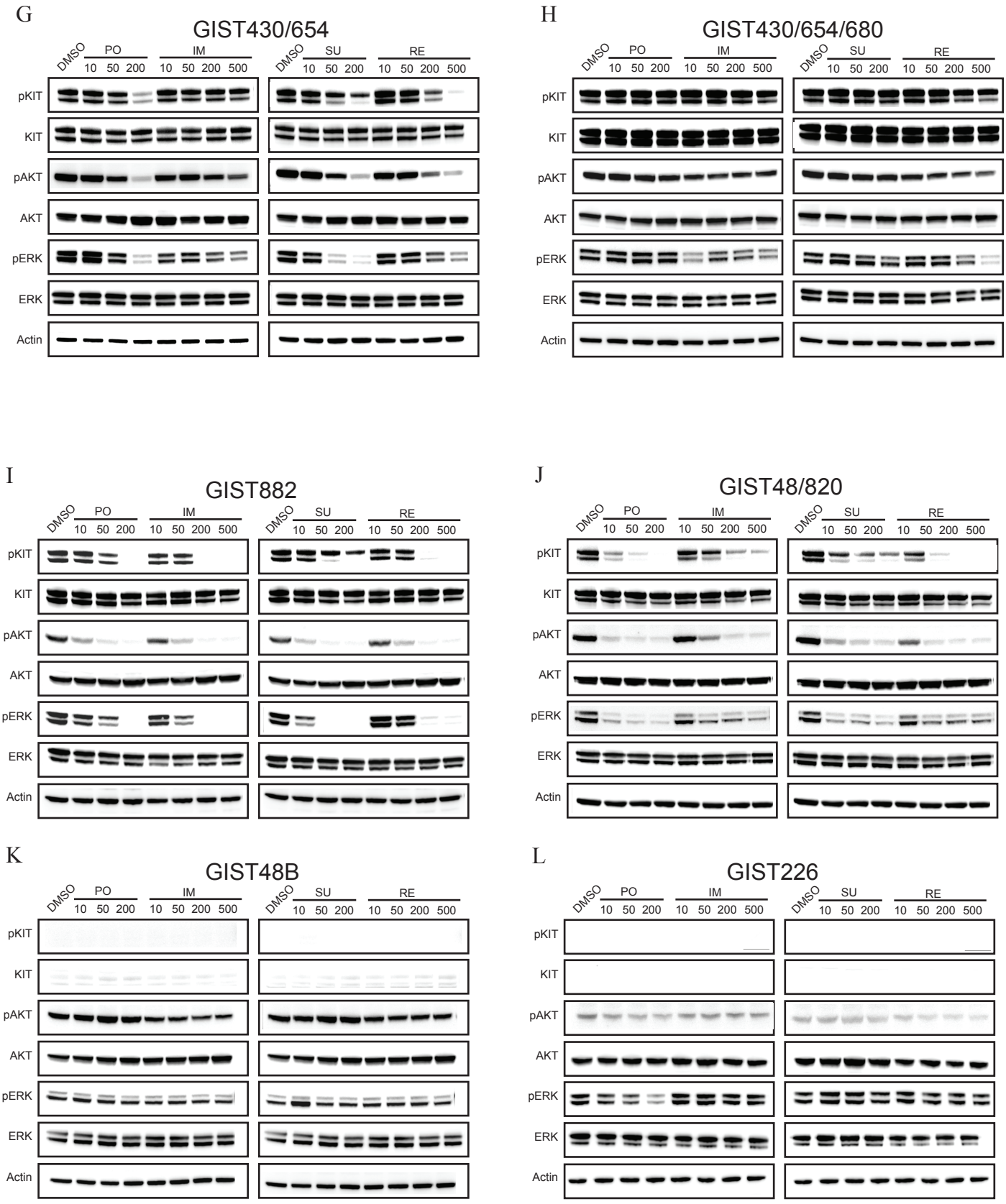


Figure S6. Impact of compound treatment on KIT signaling in GIST-derived cell lines




G
GIST430/654
H
GIST430/654/680
I
GIST882
J
GIST48/820
K
GIST48B
L
GIST226
DMSO
PO
IM
SU
RE
10 50 200
10 50 200 500
pKIT
KIT
pAKT
AKT
pERK
ERK
Actin