

Supplementary information, Figure S2 Validation of SMARCE1 in modulating drug responses to MET and ALK inhibitors.

Ectopic expression of a RNAi-resistant SMARCE1 cDNA re-sensitizes SMARCE1 knockdown cells to MET inhibition in H1993MET-amplified cells (A) or ALK inhibition in H3122 cells harboring the *EML4-ALK* translocation (**B**). (A) H1993 cells expressing pRS control or independent shSMARCE1 vectors were retrovirally infected with viruses containing pMX or pMX-SMARCE1 (RNAi resistant), and were grown in the absence or presence of 300 nM Crizotinib, 150 nM EMD1214063, or 150 nM PHA665752. Cells were then fixed, stained with crystal violet after 12 days (untreated) or 28 days (treated). Crystal violet was then extracted with 10% Acetic Acid and measured at OD 590 nM. Error bars denoate SD; *, ** and *** denote p values <0.05, < 0.01 and < 0.001 of four independent biological replicates respectively. (B) H3122 cells expressing pRS control or independent shSMARCE1 vectors were retrovirally infected with viruses containing pMX or pMX-SMARCE1 (RNAi resistant), and were grown in the absence or presence of 300 nM Crizotinib, 20 nM Ceritinib or 5 nM NVP-TAE684. Cells were then fixed, stained with crystal violet after 10 days (untreated) or 21 days (treated). Crystal violet was then extracted with 10% Acetic Acid and measured at OD 590 nM. Error bars denoate SD; *, ** and *** denote p values <0.05, < 0.01 and < 0.001 of four independent biological replicates respectively.