



Supplementary information, Figure S6 Multiple SWI/SNF components regulate

EGFR expression and drug responses to MET and ALK inhibitors in NSCLC cells.

(A) Downregulation of different SWI/SNF components all lead to upregulation of *EGFR* expression. *SMARCE1, ARID1A, SMARCA4* and *EGFR* mRNA expression levels in the H1993 cells expressing pRS control or independent shRNA vectors targeting *SMARCE1, ARID1A or SMARCA4* were measured by qRT-PCR. Error bars denote SD; \* denote p values <0.05 of independent three biological replicates.

(**B**) Suppression of *SMARCE1*, *ARID1A* or *SMARCA4* confers resistance to MET inhibitors in *MET*-amplified cells. H1993 cells described above (A) were grown in the absence or presence 300 nM Crizotinib, 150 nM EMD1214063, or 150 nM PHA665752. Cells were then fixed, stained and photographed after 12 days (untreated) or 28 days (treated).

(C) Downregulation of different SWI/SNF components lead to differential upregulation of *EGFR* expression. *SMARCE1, ARID1A, SMARCA4* and *EGFR* mRNA expression levels in the H3122 cells expressing pRS control or independent shRNA vectors targeting *SMARCE1, ARID1A or SMARCA4* were measured by qRT-PCR. Error bars denote SD; \*, \*\* and \*\*\* denote p values <0.05, <0.01 and < 0.001 of three independent biological replicates respectively; ns, not significant.

(**D**) Suppression of *SMARCE1* or *ARID1A* but not *SMARCA4* confers resistance to ALK inhibitors in EML4-ALK positive cells. H3122 cells described above (C) were grown in the absence or presence of 300 nM Crizotinib, 20 nM Ceritinib or 5 nM NVP-TAE684. Cells were then fixed, stained and photographed after 10 days (untreated) or 28 days (treated).