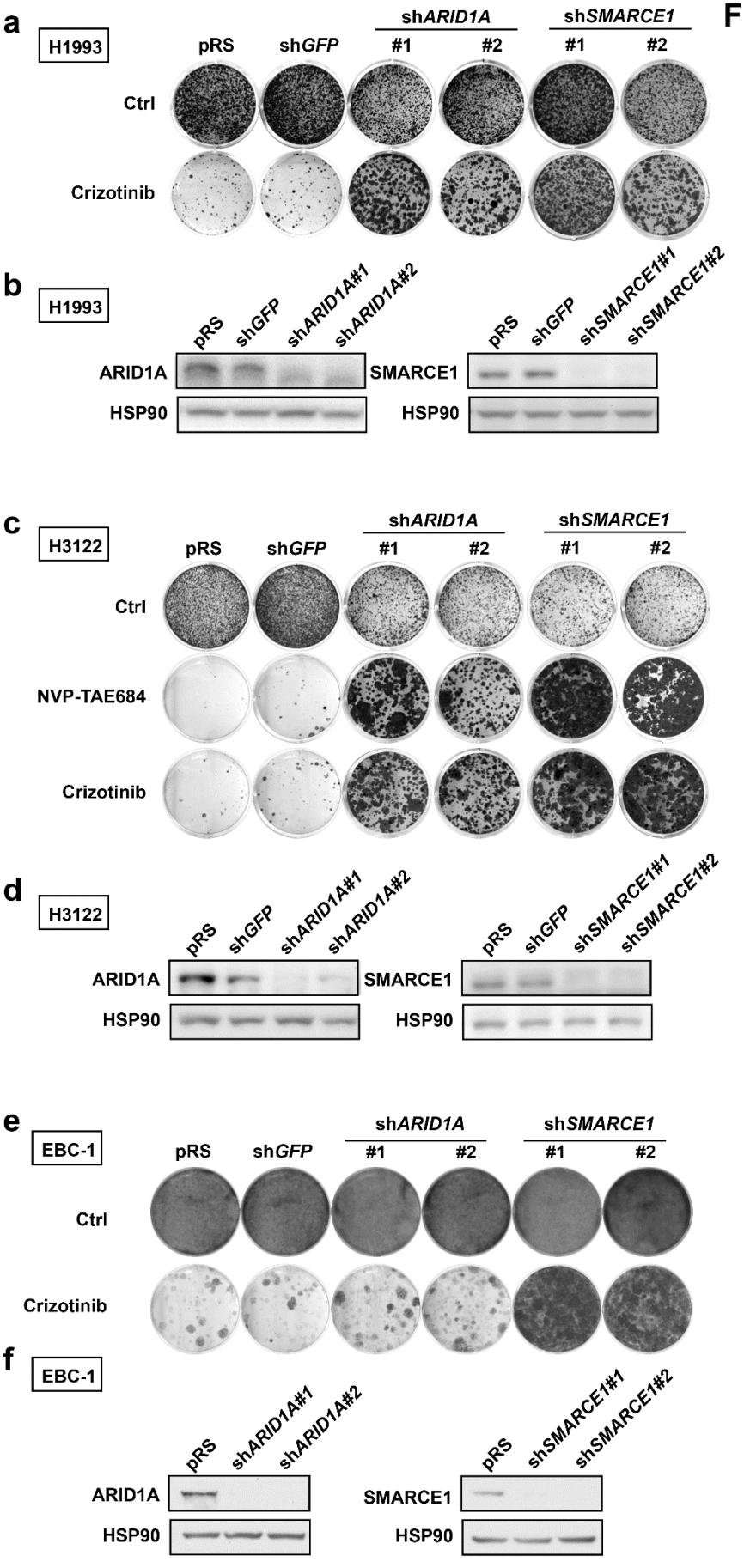
Figure S1


a
H1993
pRS
shGFP
shARID1A
#1
#2
shSMARCE1
#1
#2
Ctrl
Crizotinib
b
H1993
pRS
shGFP
shARID1A#1
shARID1A#2
ARID1A
HSP90
pRS
shGFP
shSMARCE1#1
shSMARCE1#2
SMARCE1
HSP90
c
H3122
pRS
shGFP
shARID1A
#1
#2
shSMARCE1
#1
#2
Ctrl
NVP-TAE684
Crizotinib
d
H3122
pRS
shGFP
shARID1A#1
shARID1A#2
ARID1A
HSP90
pRS
shGFP
shSMARCE1#1
shSMARCE1#2
SMARCE1
HSP90
e
EBC-1
pRS
shGFP
shARID1A
#1
#2
shSMARCE1
#1
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Ctrl
Crizotinib
f
EBC-1
pRS
shARID1A#1
shARID1A#2
ARID1A
HSP90
pRS
shSMARCE1#1
shSMARCE1#2
SMARCE1
HSP90

Supplementary information, Figure S1 Validation of individual shRNAs targeting *ARID1A* and *SMARCE1* in modulating drug responses to MET and ALK kinase inhibitors in multiple NSCLC cell systems.

(A and B) Independent shRNAs targeting *ARID1A* or *SMARCE1* confer resistance to MET inhibition in H1993 cells. (A) Expression of non-overlapping retroviral sh*ARID1A* (#1 and #2) or *shSMARCE1* vectors (#1 and #2) in H1993 cells confers resistance to MET inhibition, as indicated by long-term colony formation assays. Cells were either untreated or cultured in the presence of 300 nM crizotinib. The pRS and sh*GFP* vectors were used as negative controls. The cells were fixed, and stained with crystal violet after 14 (untreated) or 28 (treated) days. (B) The level of knockdown of ARID1A and SMARCE1 protein in the cells described in S1A was measured by western blotting. HSP90 was used as a loading control.

(C and D) Independent shRNAs targeting *ARID1A* or *SMARCE1* confer resistance to ALK inhibition in H3122 cells. (C) Expression of non-overlapping retroviral sh*ARID1A* (#1 and #2) and *shSMARCE1* vectors (#1 and #2) in H3122 cells confer resistance to ALK inhibition, as indicated by long-term colony formation assays. Cells were either untreated or cultured in the presence of 2.5 nM NVP-TAE684 or 300 nM crizotinib. The pRS and sh*GFP* vectors were used as negative controls. The cells were fixed, and stained with crystal violet after 14 (untreated) or 28 (treated) days. (D) The level of knockdown of ARID1A and SMARCE1 protein in the cells described in S1C was measured by western blotting.

(E and F) Suppression of SMARCE1 but not of ARID1A confers resistance to crizotinib in EBC1 cells. (E) Non-overlapping retroviral sh*ARID1A* (#1 and #2) and *shSMARCE1* vectors (#1 and #2) were expressed in EBC1 cells. The pRS and sh*GFP* vectors were used as negative controls. Cells were either untreated or cultured in the presence of 50 nM crizotinib. The cells were fixed, and stained with crystal violet after 10 (untreated) or 32 (treated) days. Long-term colony formation assays indicate that SMARCE1 but not of ARID1A confers resistance to MET inhibition. (F) The level of knockdown of ARID1A and SMARCE1 protein in the cells described in S1E was measured by western blotting.