Supplementary Figure 1

(a) EI 175H cells were analyzed for p53 expression upon ponA induction as assessed by Western blot. (b) Immunostaining for ZO-1 and DAPI (left and middle panels) and p53 and DAPI (right panels) in EI control (left and right panels) or EI 273H cells (middle and right panels) after ponA induction for 24 hrs. Scale bars indicate 10 μm. (c) Immunostaining for ZO-1 and DAPI (left and middle panels) and MET and DAPI (right panels) in H1299 cells that were transfected with control or MET siRNA and induced with ponA or HGF for 24 hrs. Scale bars indicate 10 μm. (d) Scattering of EI 175H cells that were transfected with MET siRNA, followed 6 hrs later by transfection with a MET expression construct, and 16 hrs later treated with ponA for 48 hrs. Scale bars indicate 50 μm. MET levels were assessed by Western blot (bottom panel) and Actin was used as loading control. (e) EI 175H cells were allowed to form colonies, then incubated with EGF for 16 hrs and analyzed for scattering using time lapse microscopy. Scale bars indicate 20 μm. (f) Immunostaining for ZO-1 and DAPI in EI 175H H1299 cells after EGF induction for 24 hrs. Scale bars indicate 10 μm.

Supplementary Figure 2

(a) Western blot analysis to detect phosphorylation of ERK1/2 in EI 175H H1299 induced with increasing doses of ponA or HGF as indicated. GCN5 and total ERK1/2 levels were used as loading control. The numbers indicate quantification of pERK1/2 levels as compared to EV transfected cells and normalized for total ERK1/2 levels. (b) Western blot analysis to detect phosphorylation of ERK1/2 in H1299 transfected with EV, 175H or 273H. GCN5 and total ERK1/2 levels were used as loading control. The numbers indicate quantification of pERK1/2 levels as compared to EV transfected cells and normalized for total ERK1/2 levels. (c) Western blot analysis to detect HGF-induced phosphorylation of ERK1/2 and MET in 273H H1299 cells that were transfected with MET siRNA or treated SU11274 (MET inhibitor) as indicated. GCN5 and total ERK1/2 levels were used as loading control.

Supplementary Figure 3

(a) EI 175H H1299 cells were analyzed for invasion capacity in fibronectin supplemented Matrigel using HGF as a chemo-attractant after induction with increasing doses of ponA (right panels). The induction of p53 was verified by Western blot and Actin was used as loading control (left panels). (b) H1299 EV and 273H cells were analyzed for invasion capacity in fibronectin supplemented Matrigel using IGF-1 or PDGF-β as chemo-attractants. (c) H1299 cells were starved for 1 hr and incubated in IGF-1 or PDGF-β as indicated, then analysed for AKT phosphorylation by Western blot. Total AKT and actin were used as loading controls. (d) Mutant p53 (273H) or control (EV) H1299 cells were analyzed for invasion capacity in fibronectin supplemented Matrigel using HGF as a chemo-attractant in the presence or absence of the EGFR inhibitor PD15305 (PD).

Supplementary Figure 4

Immunostaining for E-cadherin, p53, and DAPI (left panels) or E-cadherin, ZO-1 and DAPI (right panels) in HT29 (a) or A431 (b) cells that were transfected with control siRNA or p53 siRNA for 16 hrs, followed by HGF treatment for 16 hrs. Scale bars indicate 10 μm.

Supplementary Figure 5

(a) Invasion of EV, 273H cells or 273H cells transfected with siRNA against p63 in fibronectin supplemented Matrigel towards medium that contained no growth factors. (b) Scattering of H1299 cells 48 hrs after transfection with individual siRNAs targeting p63.

Supplementary Movie 1

Serial section fluorescent pictures of H1299 273H cells invading towards EGF (a) or HGF (b) were captured and reconstructed into 3D views. Movies were generated to show branched structures in the 273H cells invading towards HGF.