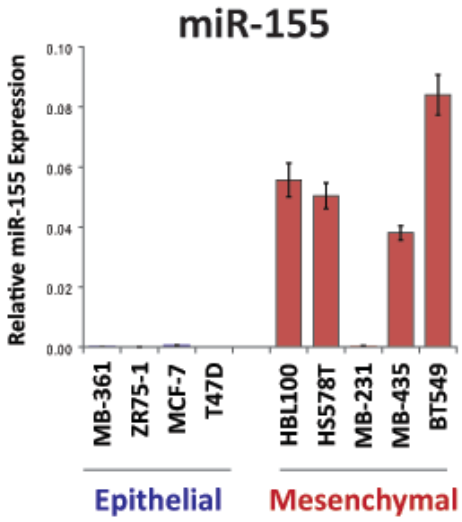
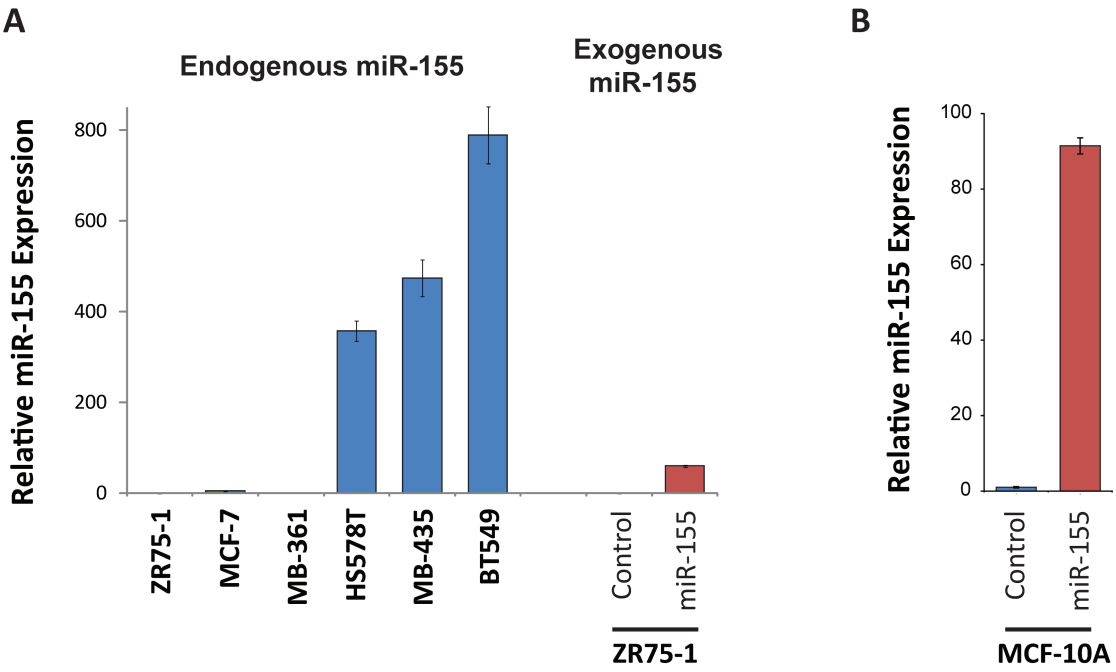
Supplementary Figure 1



Supplementary Figure 1. miR-155 expression in breast cancer cell lines

The relative expression levels of miR-155 were determined in a panel of epithelial and mesenchymal breast cancer cell lines (as indicated).

Supplementary Figure 2

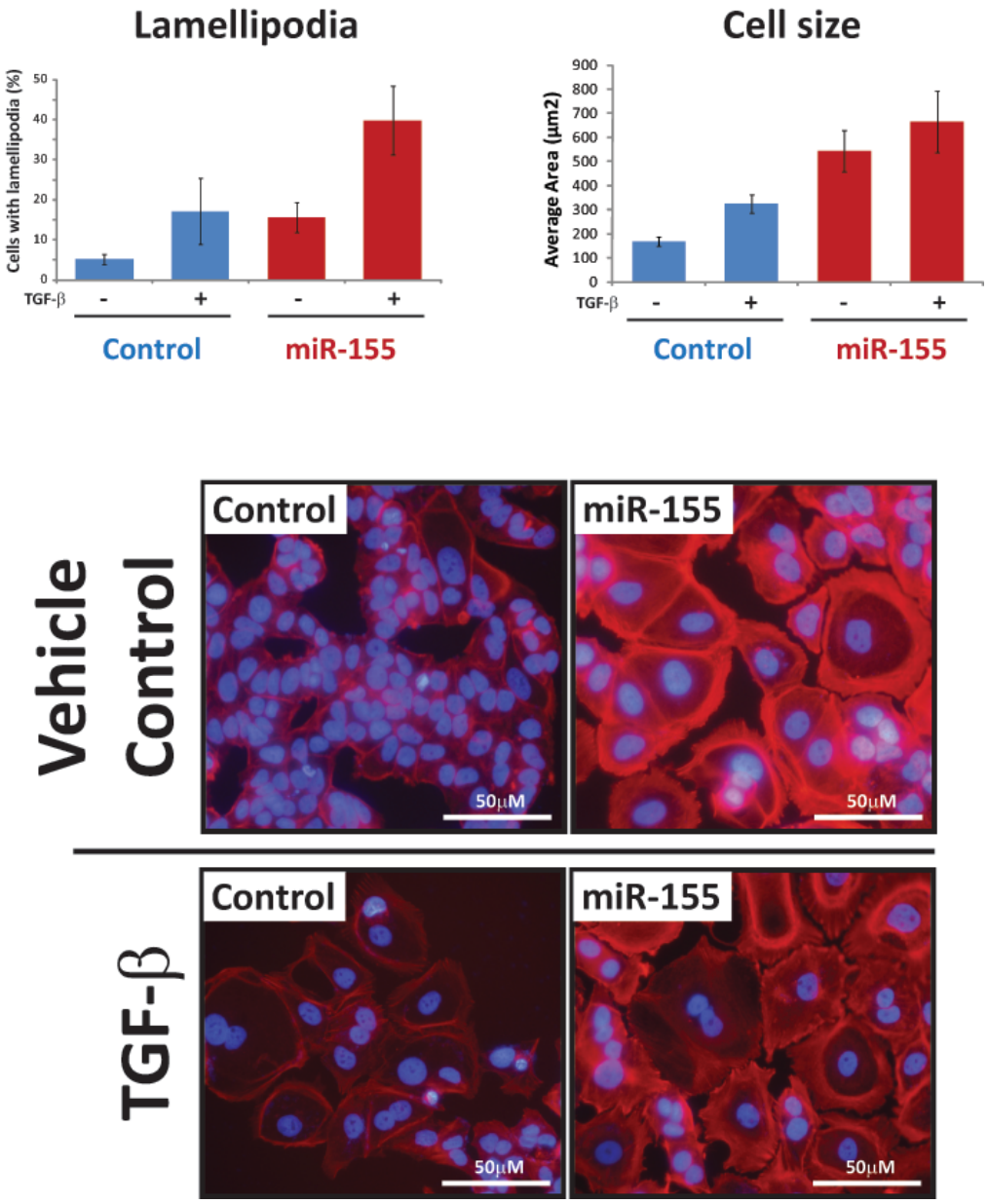


Supplementary Figure 2. Physiologically-relevant expression of miR-155 in ZR-75-1 cells

A. The relative expression levels of miR-155 were determined in a polyclonal population of ZR-75-1 cells stably expressing either retrovirally-delivered miR-155 or a scrambled non-targeting RNA, as compared with a panel of epithelial and mesenchymal breast cancer cell lines.

B. Relative expression of miR-155 was determined in polyclonal populations of MCF-10A cells stably expressing either retrovirally-delivered miR-155 or a scrambled non-targeting RNA.

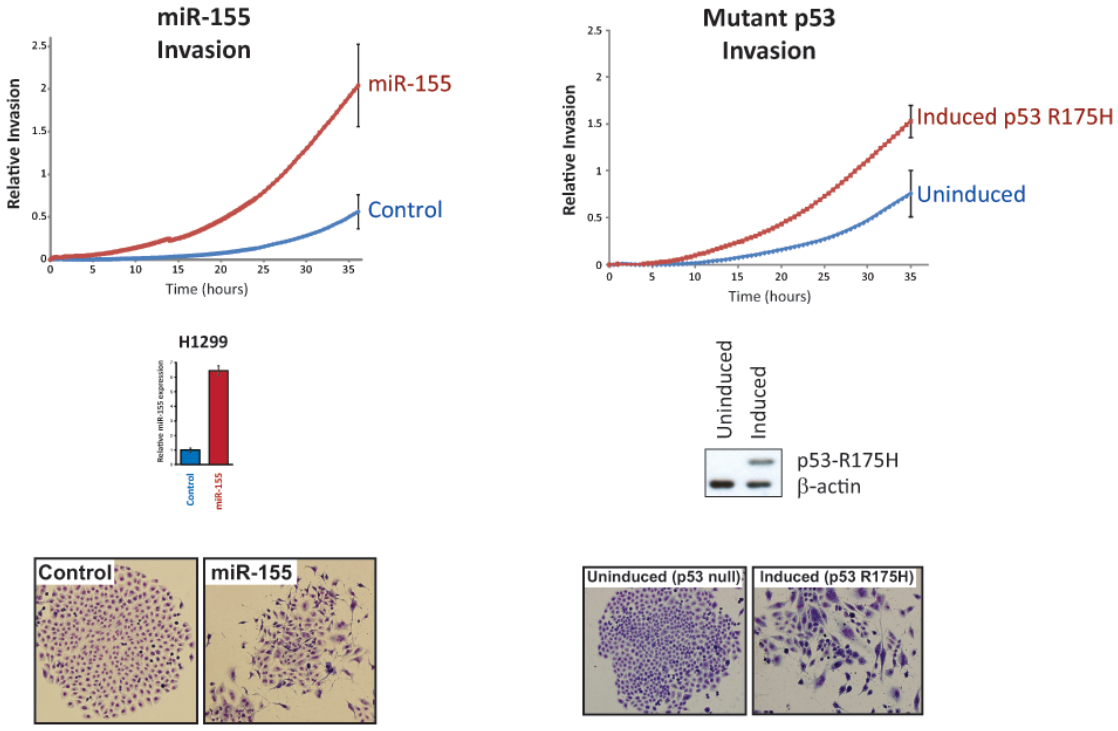
Supplementary Figure 3



Supplementary Figure 3. miR-155 expression phenocopies activation of the TGF-β pathway.

ZR-75-1 cells (control or miR-155) were seeded at 10% confluence and grown for 12 days in the presence of 2 ng/mL TGF-β (or vehicle control). Cells were stained for F-actin using a phalloidin antibody, with lamellopodia and cell size scored as described in Materials and Methods.

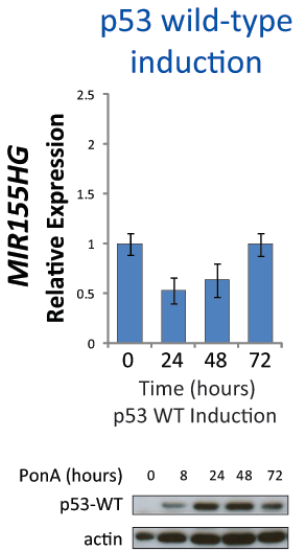
Supplementary Figure 4



Supplementary Figure 4. miR-155 expression phenocopies mutant p53 expression in H1299 cells.

H1299 cells expressing either miR-155 or a scrambled non-targeting RNA were plated in the upper chamber of a CIM-16 plate coated with 5% matrigel, with invasion assessed in real-time using an Xcelligence RTCA DP analyser. Similar experiments were performed using EI H1299 p53 R175H cells in the presence or absence of the inducing agent, PonA (2.5 µg/mL). The relative expression of miR-155 and p53 R175H were determined by real-time PCR and Western blot analysis, respectively. H1299 cells (as treated above) were plated in a 6-well format at 1000 cells/well and stained with Giesma after 10 days. Pictures show representative colonies.

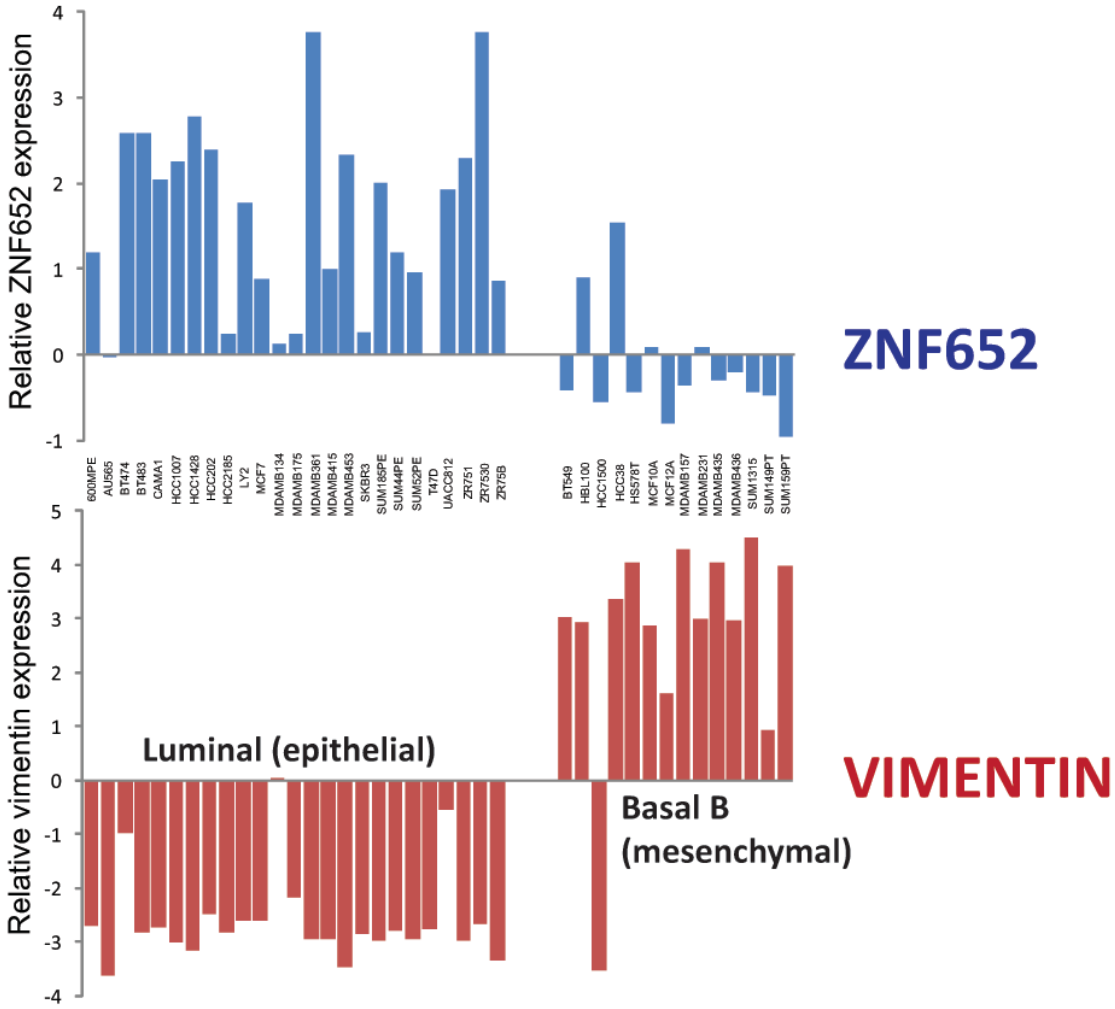
Supplementary Figure 5



Supplementary Figure 5. miR-155 is directly repressed by wild-type p53

EI p53-WT cells were treated with 2.5 µg/mL PonA for 0, 24, 48 or 72 hours to induce p53 protein expression. Relative *MIR155HG* expression and p53-WT protein expression were determined by specific RT-PCR or Western blot analysis, respectively.

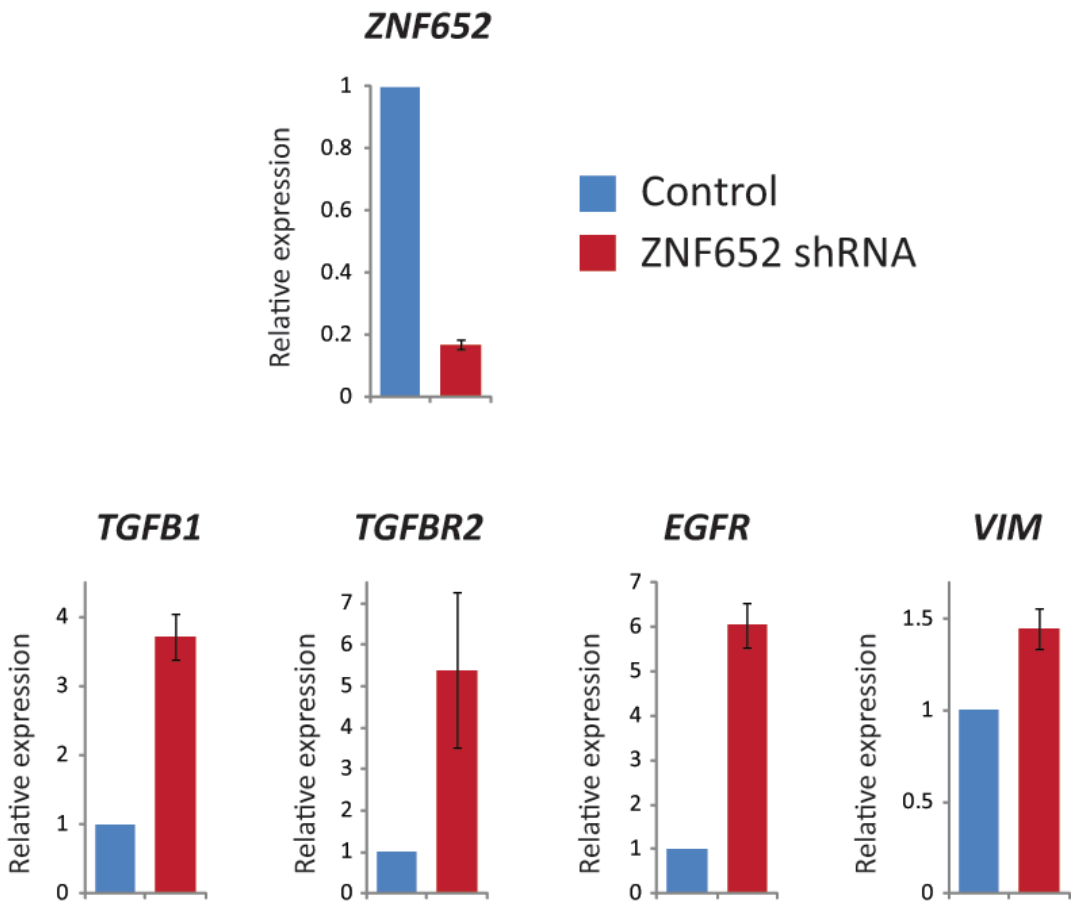
Supplementary Figure 6



Supplementary Figure 6. ZNF652 expression is inversely correlated with vimentin in breast cancer cell lines

Expression microarray data from a cohort of 51 breast cancer cell lines representing both luminal (epithelial) and basal b (mesenchymal) cell types (Neve, 2006) shows that ZNF652 expression is up-regulated in epithelial cell lines and inversely correlated with the expression of the mesenchymal marker, vimentin.

Supplementary Figure 7



Supplementary Figure 7. Knockdown of ZNF652 results in a de-repression of target genes

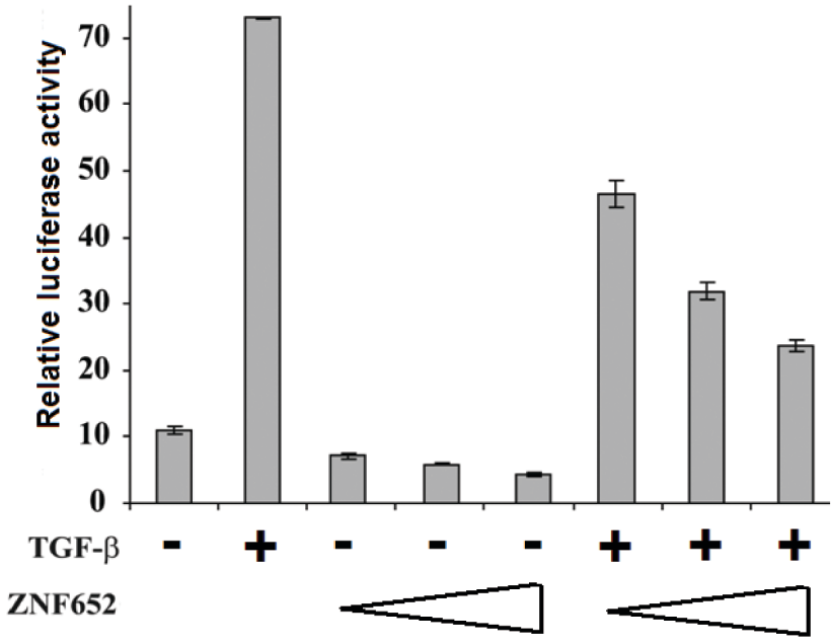
Polyclonal populations of ZR-75-1 cells expressing a ZNF652 shRNA (V3LHS_368392) or non-targeting (control) sequence were generated using the pGIPZ lentiviral shRNAmir system. The expression of ZNF652 and its target genes (*TGFB1, TGFBR2, EGFR and VIM*) were determined using real-time PCR. Similar results were observed using a second ZNF652-specific shRNAmir construct (V2LHS_229362) (data not shown).

Supplementary Figure 8

Supplementary Figure 8. miR-155 and ZNF652 regulate similar target genes within the EMT pathway.

The expression of 84 EMT related genes (listed in Supplementary Table 1) was determined in ZR-75-1 cells with silenced ZNF652 expression (from Supplementary Figure 7). Data presented is an average of three independent biological replicates. Heat maps show the comparison of these data with that of ZR-75-1 cells expressing miR-155 (from Figure 1E).

Supplementary Figure 9



Supplementary Figure 9. ZNF652 suppresses SMAD-mediated transactivation

H1299 cells were transiently-transfected with ZNF652 (25, 100 or 250 ug) and a firefly luciferase reporter construct driven by a canonical SMAD response element. Cells were incubated in the presence of 2 ng/mL TGF-β (or vehicle control) where indicated. Firefly luciferase activity was determined using a dual-luciferase reporter kit and normalised against a renilla luciferase internal transfection control as described in Materials and Methods.