Supplemental Figure legends

Fig. S1. Direct targeting of ABCC5 and Bmi-1 by miR-128 in SK-3rd and MCF-7 cells.

(A) SK-3rd adherent cells and (B) MCF-7 adherent cells, which expressed significantly more miR-128 compared with their mammospheres, were transfected with 100 nmol/L miR-128 inhibitor or scrambled oligonucleotide as a negative control. Each experiment was performed in triplicate. Expression of luciferase with the putative miR-128 target site in wild-type (wt) or mutated 3'UTR from ABCC5 or Bmi-1 was measured in a luminometer and is shown as a fraction of control. (**, $P<0.001$ vs. untransfected control). (C) Semi-quantitation of immunoblotting with anti-Bmi-1 and anti-ABCC5 antibodies demonstrates that Bmi-1 and ABCC5 proteins are elevated in mammospheres compared with their differentiated cells in SK-3rd, MCF-7 cells or primary cells. (**, $P<0.001$ vs. adherent cells). (D) Semi-quantitation of immunoblotting demonstrates that Bmi-1 and ABCC5 proteins decrease with the ectopic expression of miR-128 in mammospheric SK-3rd and MCF-7 cells. Transfection of lenti-Bmi-1-shRNA or lenti-ABCC5-shRNA in the BT-ICs, compared with untransfected or empty vector–transfected, led to the reduction in the expression of BMI-1 or ABCC5, respectively. (**, $P < 0.001$ vs. untransfected or empty vector–transfected cells).

Fig. S2. Cell viability of SK-3rd BT-ICs upon transfection with pMSCV-Bmi-1-del and miR-128 mimics is significantly increased under chemotherapy

Retrieving Bmi-1 expression by transfection with pMSCV-Bmi-1-del in SK-3rd BT-ICs increased the viability of SK-3rd BT-ICs treated with doxorubicin at concentrations of 4 μg/ml, 8 μg/ml and 10 μg/ml. Co-expression of ectopic miR-128 and pMSCV-Bmi-1-del, in which a

miR-128 targeting sequence is deleted, increased cell viability under the pressure of doxorubicin at concentrations of 4 μg/ml, 8 μg/ml and 10 μg/ml. (*, $p<0.01$ vs. untransfected control).

Fig. S3. Cell viability of primary BT-ICs upon transfection with miR-128 mimics is significantly reduced under chemotherapy

Mammospheres of primary cells that were obtained from breast cancer tissues were more resistant to chemotherapy when treated with doxorubicin compared with adherent cells. However, the cell viability of primary BT-ICs decreased clearly upon transfection with miR-128 mimics (*, $p<0.01$ vs. untransfected control).

Fig. S4. The higher the miR-128 level in breast tumor tissues before chemotherapy, the larger are changes in the Ki67 index or TUNEL index after chemotherapy.

Breast tumors with low miR-128 expression demonstrated a stable Ki67 index and TUNEL index following chemotherapy, while those with high miR-128 showed a dramatically reduced Ki67 index and elevated TUNEL index after chemotherapy. With the increase in miR-128 level, the changes in the Ki67 index (A) or TUNEL index (B) gradually increased after chemotherapy (% change = (after chemo–before chemo)*100/before chemo). Black circles represents patients with a complete clinical response (CR), gray circles represents partial response (PR), white circles represents stable disease (SD), and crossed circles represents progressive disease (PD).