**Supplementary material**

**Figure S1. ARID1A loss activates PI3K/AKT and has no effect on MAPK/ERK signaling in BT474 cells**

(A) BT474 cells expressing sh*ctrl* or sh*ARID1A* were cultured in the absence or presence of increasing amounts of AZD8055 for 2 hours. Cell lysates were harvested for Western blotting analysis and probed with the indicated antibodies. (B) Western analysis of two BT474 ARID1A ko clones (#2 and #20) generated with CRISPR/Cas9 targeting. Lysates were prepared three days after trastuzumab (2µg/ml) exposure and probed for the indicated proteins. (C) BT474 control cells expressing only Cas9 (ctrl) and two ARID1A CRISPR/Cas9 ko clones (#2 and #20) were cultured in the absence or presence of 2 µg/ml trastuzumab. The cells were fixed, stained and photographed after 21 days.

**Figure S2. Suppression of ARID1A leads to induction of *ANXA1* RNA expression and loss of BRG1 complex binding to *ANXA1* region.**

(**A**) BT474 and MCF7 cell lines stably infected with sh*ctrl* or two independent sh*ARID1A* vectors were subjected to RNA sequence analysis. Shown are those genes whose expression was altered 1.75 folds as a consequence of *ARID1A* suppression in both cell lines and with all tested sh*ARID1A* vectors. (**B**) BRG1 binding to the ANXA1 promoter region is diminished upon ARID1A loss. Snapshot derived from the Integrative Genomics Viewer (IGV 2.3.40) showing the Igg control and BRG1 CHIP seq data in wt MCF7 cells and a MCF7 ARID1A low subclone generated by CRISPR/Cas9 targeting. (**C**) *ARID1A* and *ANXA1* mRNA expression analysis (relative to GAPDH) by qRT-PCR in MCF7 wt cells and ARID1A low cells generated by CRISPR/Cas9 targeting.

**Figure S3. ARID1A loss correlates with ANXA1 expression in endometrial cancer**

(**A**) Boxplot of *ANXA1* TCGAmRNA expression levels for endometrial patient tumors divided in *ARID1A* wildtype (n=156) and ARID1A mutant (n=82) tumors.

(**B**) Boxplot of *ANXA1* TCGARPPA protein levels for endometrial patient tumors divided in *ARID1A* wildtype (n=116) and ARID1A mutant (n=67) tumors.

(**C**) ARID1A immunohistochemistry (IHC) inversely correlates with ANXA1 IHC in endometrial cancer. Based on nuclear staining intensity for ARID1A on whole sections, the samples were divided into ARID1A normal (moderate and strong positive, n=19) and ARID1A loss (no detectable nuclear staining, n=25). Cytoplasmic ANXA1 was scored as low (no or very weak staining), moderate or strong.

**Figure S4 Exogenous ANXA1 does not confer trastuzumab resistance**

(A) BT474 cells stably transduced with a LX304-ANXA1 vector (LX-ANXA1) were subjected to Western blot analysis. Lysates were prepared three days after trastuzumab (2µg/ml) exposure and probed for the indicated proteins. (B) BT474 cells expressing only empty vector (ctrl) and BT474 cells stably expressing the LX304-ANXA1 vector (LX-ANXA1) were cultured in the absence or presence of 2 µg/ml trastuzumab. The cells were fixed, stained and photographed after 21 days.

**Figure S5 METABRIC Boxplot and survival figure**

**(**A) *ANXA1* gene expression distribution expression in the METABRIC dataset according to pam50 subtypes. (**B-F**) Kaplan-Meier plots of the effect of the *ANXA1* gene expression in the METABRIC dataset in all (**B**) patients or according to the pam50 subtype. (**C**) Basal, (**D**) HER2, (**E**) Luminal A and (**F**) Luminal B.