**Supplementary Figure Legends**

**Supplementary Figure S1: Chromatin regulators included in this study.** A list of 551 chromatin regulators included in this study.

**Supplementary Figure S2: A chromatin regulator gene panel distinguishes docetaxel high-responders and poor-responders. A.** Correlation—as measured using R2 value—between predicted and measured tumor growth inhibition (TGI) for 21 TNBC PDX models treated with docetaxel using Random Forest classification (RF, blue) and random generalized linear model (RGLM, red). The number of genes in each panel is indicated. Random indicates a random 19-chromatin regulator gene panel and the R2 value here represents the average across 3 random, 19-chromatin regulator gene panels. **B**. Receiver operating characteristic (ROC) curves corresponding to 100 random, 19-member chromatin regulator gene panels to classify docetaxel high-responders and poor-responders. Colored bold line indicates the average of the 100 random gene panels. Bold grey line indicates the described gene panel revealed with our approach. Color indicates the cut-off threshold for classification. **C**. Clustering of 19 PDX models according to the expression of the 19 chromatin regulator gene panel and the response of the PDX models to a combination of Adriamycin (doxorubicin) and cyclophosphamide (noted: “AC”). **D.** Clustering of 15 organoids according to the expression of 10 chromatin regulators from the 19 chromatin regulator gene panel (HDAC7, SOX18, HDAC5, MBD6, MED13, RBBP4, SOX5, MCM4, RAD21, and MBD4) and the response to docetaxel. **E.** Clustering of 15 organoids according to the expression of 10 chromatin regulators from 19 chromatin regulator gene panel and the response to Irinotecan.

**Supplementary Figure S3: Comparison of Affymetrix and NanoString gene expression data. A.** Gene expression values for 8 PDX models for all chromatin regulators (blue) and the 19 chromatin regulator gene panel (red). CR: chromatin regulators

**Supplementary Figure S4:** **A chromatin regulator gene panel can distinguish response in HER2+ patients treated with docetaxel. A**. Boxplot of scaling factors used to normalize the Institut Curie cohort (Wilcoxon rank sum test; p=0.32) **B.** Boxplot of the RNA expression of the proliferation marker MKI67 in the Institut Curie responders and non-responders (Wilcoxon rank sum test; p=0.88). **C**. Boxplot of the RNA expression of HR (sum of ESR1 and PGR) in the Institut Curie high-responders and poor-responders (Wilcoxon rank sum test; p=0.74). **D**. Clustering of 23 patients according to the expression of the 18 chromatin regulator gene panel and the tumor stage. The corresponding patient response is noted. **E.** Receiver operating characteristic (ROC) curves corresponding to 100 random 18-member chromatin regulator gene panels to classify the patient response. Color indicates the cut-off threshold for classification. **F**. A histogram of AUC values for 100 random 18-member chromatin regular gene panels to predict patient response. The red line marks the AUC value for the 18 chromatin regulator gene panel revealed using our approach. The highest AUC value for a random 18-member gene panel is 0.811. resp. = responders; non-resp. = non-responders.

**Supplementary Figure S5: The SWI/SNF chromatin remodeler is associated with taxane response. A.** A focused analysis of the mechanisms leading to HLTF inactivation in the NCI-60 based on mRNA expression and promoter methylation. The x-axis shows z-scored mRNA expression and y-axis shows average promoter methylation for HLTF. Dots represent individual cell lines from the NCI-60. Cell lines with HLTF inactivation are indicated as grey dots. The Illumina Infinum HumanMethylation450 BeadChip array produced the genome-wide methylation data for all NCI-60 cell lines. Selected probes were in CpG islands within 200 base pairs of the transcription start site. The average methylation for those probes corresponds to the methylation level for the genes. **B.** HAP1 cell lines wildtype for SWI/SNF (HAP1-WT), and knockout for SMARCA4 (SMARCA4-KO) treated with a docetaxel and increasing concentrations of PFI-3, a SMARCA2/4 bromodomain inhibitor (37). The SMARCA2-KO follows a similar trend as the SMARCA4-KO (data not shown). **C.** Western blot analysis in HAP1 WT for SWI/SNF (WT), SMARCA4-KO, and SMARCA2-KO of several SWI/SNF components. **D.** The viability of HAP1 wildtype (WT) cells, SMARCA4-KO, and SMARCA2-KO after 8 days of varying concentrations of paclitaxel. The fraction of viable cells is plotted versus the paclitaxel concentration. **E.** Cell lines from the NCI-60 with a reported paclitaxel (NSC-125973) response are included. Alterations of SWI/SNF genes are represented by colored squares. The columns are organized first by paclitaxel sensitivity and then by tissue of origin (color-coded) where BR: breast, CNS: central nervous system, CO: colorectal, LE: leukemia, LC: lung, OV: ovarian, RE: renal. The rows present the SWI/SNF genes ranked by alteration frequency in the NCI-60.

**Supplementary Table S1: Clinical characteristics of the Institut Curie patient cohort.** For more information, see Materials and Methods.