**Supplementary Figures Legends**

**Supplementary Fig. 1** Study flow diagram of patient cohorts

**Supplementary Fig. 2** Distributions of signature scores BRCAness and TIS (**A, B**) and expression values of AR (**C**) in the verification cohort (BreastMark, black) were adjusted to the discovery cohort (IKP211, grey) by median-centering. No difference between the distributions of both cohorts was observed (Wilcoxon test).

**Supplementary Fig. 3.** Event-free survival (EFS) stratified by PAM50 subtype across all IKP211 patients (N=612). The majority of patients were of Luminal A (LumA, 58.5%), followed by Luminal B (LumB, 32.7%), HER2 (6%), and Basal (2.8%) subtypes. Patients were categorized as lower-risk (LumA) and higher-risk (LumB, HER2, Basal) for subsequent analyses based on pairwise hazard ratios using LumA as a reference.

**Supplementary Fig. 4.** Pairwise spearman correlation matrix between signatures derived in patients from the discovery cohort (IKP211). Colors refer to positive (red) or inverse (blue) correlation coefficients, pairwise comparisons with significant correlations following Holm-Bonferroni correction for multiple testing are indicated by stars.

**Supplementary Fig 5.** Confirmation of an association between gene expression scores and event-free survival (EFS) in the BreastMark cohort. Kaplan-Meier curves and corresponding Log rank *P* values are shown for subgroups of patients with the number of events (ev) in brackets. Expression scores in the independent confirmation cohort were centered to the median of the IKP211 cohort and cutoffs identified in IKP211 were applied. **A**. Higher-risk patients, **B.** Lower-risk patients, stratified by AR expression, respectively. **C.** Higher-risk patients, **D.** Lower-risk patients, stratified by BRCAness signature, respectively.

**Supplementary Fig. 6.** Consensus clustering analysis of mean-centered and standardized signature scores across all IKP211 patients. Four major groups across BC360 signatures (columns) were observed, of which immune-activity signatures including TIS are grouped within cluster A. Group B is composed of pathways related to tumor responsiveness and regulation including the BRCAness signature, group C combines steroid receptor signaling signatures including the AR, and the remaining signatures form a heterogenous cluster D. Hierarchical clustering of the patients (rows) revealed a distinct patient group comprising the majority of patients with high TIS >7.52 (left column, red tips). Heatmap colors indicate low (blue) to high (red) standardized score expression.

**Supplementary Fig. 7.** Examples of HE-sections of tumors examined for the number of tumor infiltrating immune cells (TIL). **A.** 1%; **B.** 5%; **C.** 40%; **D.** >60% TILs. Tumor stroma was evaluated for the presence of TILs by quantitating the proportion of stroma area infiltrated by immune cells (0%-100%; (28)). Counts were rounded to multiples of 5%. Immune cells, predominantly T-lymphocytes are indicated by red arrows.

**Supplementary Fig. 8.** Association between the number of tumor infiltrating immune cells (TIL) inferred by histological examination of tumor sections and gene expression signatures reflecting cell abundances of cytotoxic cells (left) and CD8+ T-cells (right). Tumor stroma was evaluated for the presence of TILs by quantitating the proportion of stroma area infiltrated by immune cells (0%-100%; (28)). Counts were rounded to multiples of 5%. Correlation coefficients and *P* value refer to Pearson’s correlation test.