**Supplementary Methods and Analysis**

*Gene expression data*

RNA was extracted from multiple FFPE cores of 1 or 1.5 mm taken from pathologists’ annotated regions of tumor (n=1-3 cores) or histologically normal tumor-adjacent tissue (n=3-5 cores). Tumor-adjacent tissue was obtained greater than one centimeter from the invasive carcinoma whenever possible; a minimum of approximately 2 mm between tumor and histologically normal tumor-adjacent tissue was permitted. Selected areas of tumor-adjacent normal tissue were required to be truly histologically-normal appearing (i.e. no lesser degrees of atypia or proliferative/metaplastic alterations were permitted in the area designated normal). RNA was extracted using the AllPrep DNA/RNA FFPE Kit (Qiagen, Valencia, CA); 50ng of RNA was sent to the microarray facility. Gene expression data were obtained using microarrays performed in 2012-2014 and 2015-2018 by the Molecular Biology Core Facilities, Dana-Farber Cancer Institute, Boston, MA. The microarrays between 2012 and 2014 were performed using the WT-Ovation® FFPE WTA System amplification kits (NuGEN, San Carlos, CA) and Glue Grant Human Transcriptome Arrays (HTA 3.0 pre-release version; Affymetrix, Santa Clara, CA). Microarrays between 2015 and 2018 were performed using the Genisphere® Sensation™ (NuGEN) or GeneChip® WT Pico (Affymetrix) amplification kits and Affymetrix HTA 2.0 arrays. All microarrays were scanned with the GeneChip® Scanner 3000 7G (Affymetrix).

Gene expression data were normalized, summarized into log2 values using Robust Multi-array Average (RMA) and annotated. Sample quality was evaluated using Affymetrix Power Tools probeset summarization based metrics, including the area under the curve (AUC) that plots the detection of positive control probes against the false detection of negative controls. Due to the lower quality nature of FFPE samples, samples with AUC of >0.55 were retained and another round of quality control using arrayQualityMetrics (2012-2014 v3.24.0; 2015-2018 v3.32.0) was carried out. In total, 1577 microarrays from 954 women passed quality control. All microarrays and sample information are available at the National Center for Biotechnology Information Gene Expression Omnibus (accession number: GSE115577).

*Molecular subtyping by PAM50 using the modified median gene centering pre-processing method*

This process was carried out separately for tumor and tumor-adjacent samples. The original training set in Parker *et al.* contained equal distribution of ER+ and ER- tumor samples. Thus, the ER distribution of the intended research cohort (i.e., NHS/NHSII) ought to match the original training set in Parker *et al*. For tumor samples, we first created 100 random subsets where each subset contained 50% ER+ and 50% ER- samples. The average of 100 median expressions for each PAM50 gene was obtained. Modified median gene centering was performed by subtracting the calculated median expression for each PAM50 gene from all tumor samples. Research-based PAM50 subtyping was carried out adhering to Parker *et al*.For tumor-adjacent tissues, the median adjustment factors were calculated from tumors samples.

*Molecular subtyping by PAM50 using the subgroup-specific gene centering pre-processing method*

Likewise, this process was carried out separately for tumor and tumor-adjacent samples. The median value for each PAM50 gene was calculated using the original Parker *et al.* training set (*n*=189)*.* One hundred random subsets of the original training data were created to match the ER distribution in the NHS/NHSII cohorts (2012-2014: 78% ER+ among tumor samples and 80% ER+ among tumor-adjacent; 2015-2018: 85% ER+ among tumor samples and 82% ER+ among tumor-adjacent). Within each random subset, the percentile that corresponded to each PAM50 gene’s median value was obtained; the average of 100 percentiles was calculated. The expression value for each PAM50 gene corresponding to its average percentile in the NHS/NHSII cohorts was identified. Subgroup-specific gene centering was carried out by subtracting the identified gene expression value from all NHS/NHSII samples and research-based PAM50 classification was performed.

*Proliferation and ROR-PT scores*

The PAM50 proliferation score is the average expression of 11 proliferation-related genes that are part of the PAM50 gene signature: *BIRC5, CCNB1, CDC20, CEP55, MKI67, NDC80, NUF2, PTTG1, RRM2, TYMS* and *UBE2C*. ROR-PT scores and groupings (low, medium and high) were automatically generated after modified median or subgroup-specific gene centering pre-processing.

*Molecular subtyping using IHC surrogates*

IHC data were obtained from tissue microarrays containing three cores per individual. The maximum expression across cores was utilized for all markers. ER+ and PR+ were defined as the presence of any nuclear staining (>1%). HER2+ was defined as strong cell membrane staining in >10% tumor cells, and over-expression of marker of proliferation Ki-67 protein (Ki-67) was defined as nuclear staining in >14% of tumor cells. CK 5/6+ and EGFR+ were defined as the presence of any cytoplasmic and/or membrane stains of tumor cells. Missing IHCs for ER, PR and HER2 (*n*=144) were replaced with data from medical records. For tumors missing Ki-67 IHC data (*n*=545), histologic grade was used as a proxy. Tumors were classified into Luminal A, Luminal B, HER2-enriched and Basal-like as previously defined. Luminal A was defined as ER+ and/or PR+, HER2-, and Ki-67 low (or histologic grade 1 or 2). Luminal B was defined as ER+ and/or PR+, and HER2+; or ER+ and/or PR+, HER2-, and Ki-67 high (or histologic grade 3). HER2-enriched tumors were ER-, PR- and HER2+ while Basal-like tumors were ER-, PR-, HER2-, and CK 5/6+ and/or EGFR+. Tumors were “unclassified” if they were ER-, PR-, HER2-, CK 5/6- and EGFR- (*n*=16). Tumors were “missing” if they had incomplete data necessary for classification (*n*=168).