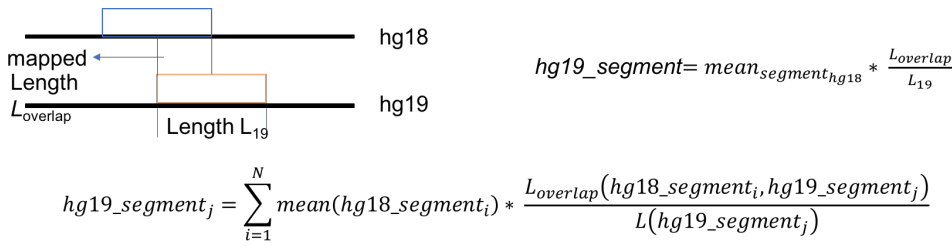


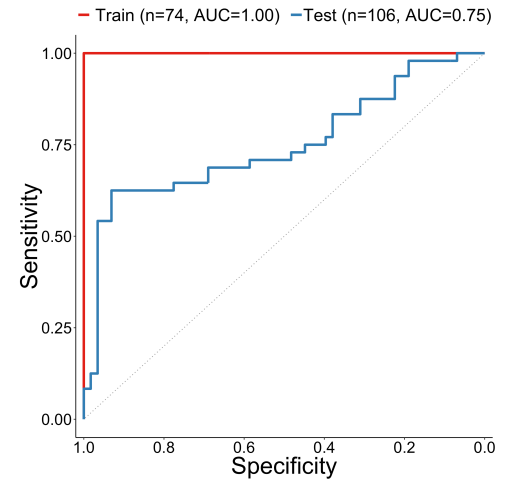
**Supplemental Figure 1. Details of SVM BRCA1-like classifier. (A)** Overview of copy number mapping algorithm for generating the input for training the SVM BRCA1-like classifier. **(B)** Receiver-operation characteristic curves (ROC) of the classifier applied to training and test set (AUC=1.00 and 0.75, respectively). **(C-D)** Correlation of SVM BRCA1-like probability scores with published HR-deficiency metrics (HRD-LOH and LST scores). \*\*\*  $P < 0.001$ .

**(A)**

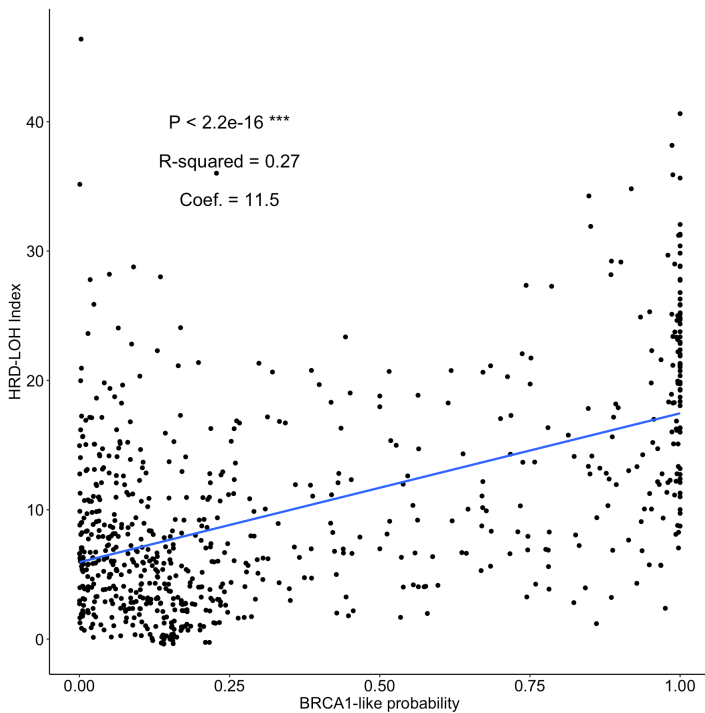


where  $hg19\_segment_j$  is the  $j$ -th segment in the hg19 reference assembly,  $N$  is the number of segments in the hg18 assembly that are mapped onto hg19, and  $hg18\_segment_i$  is the  $i$ -th mapped segment in hg18.

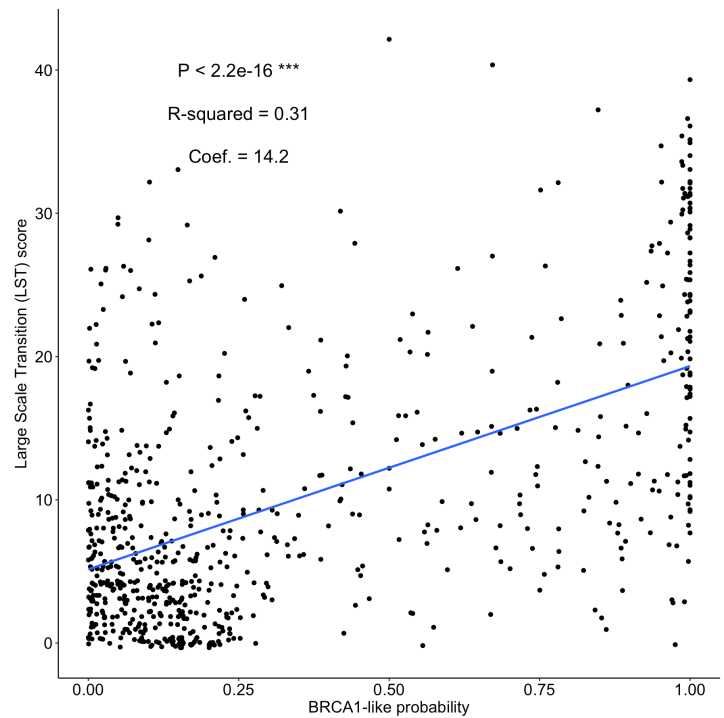
**(B)**



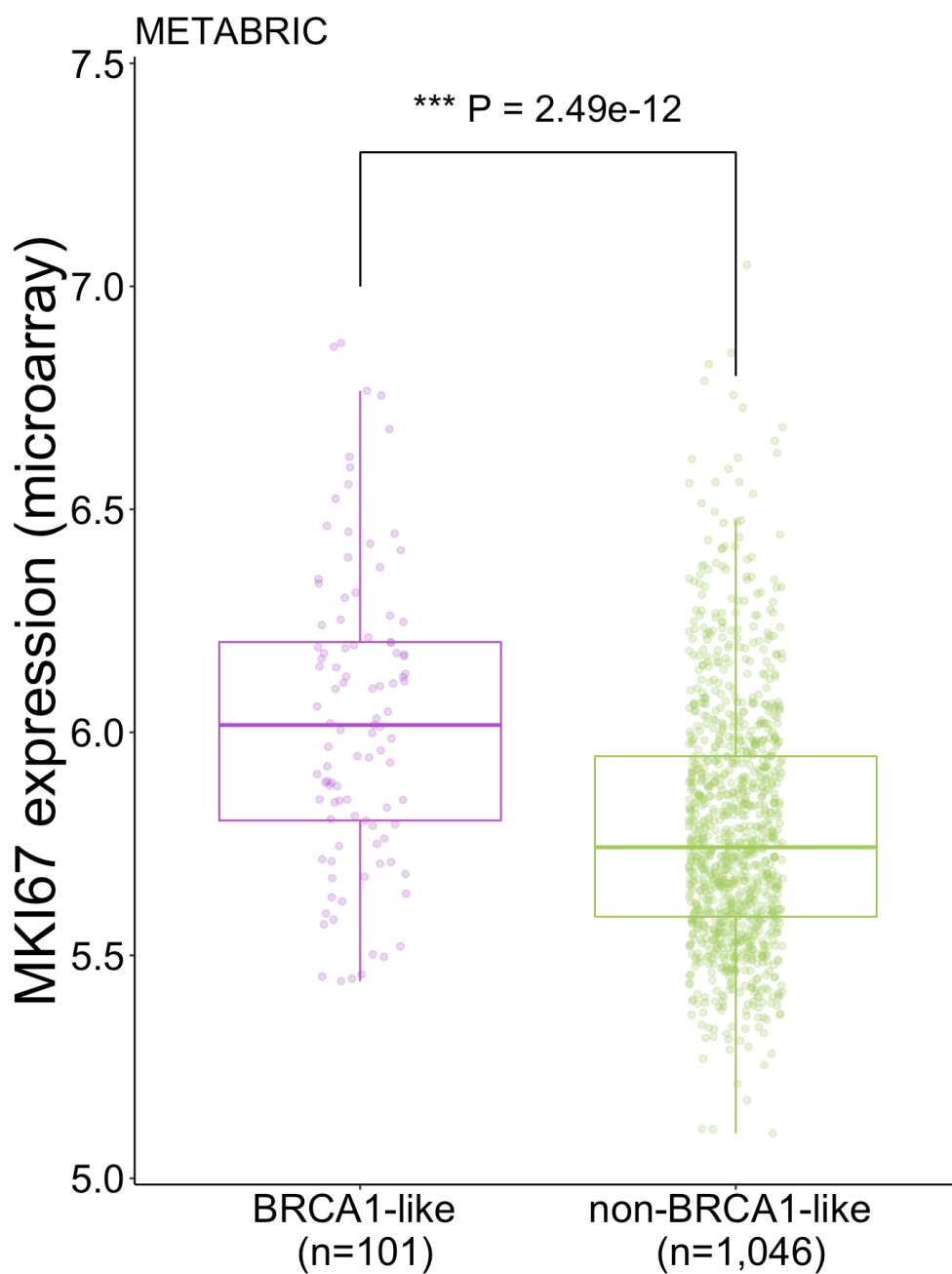
**(C)**



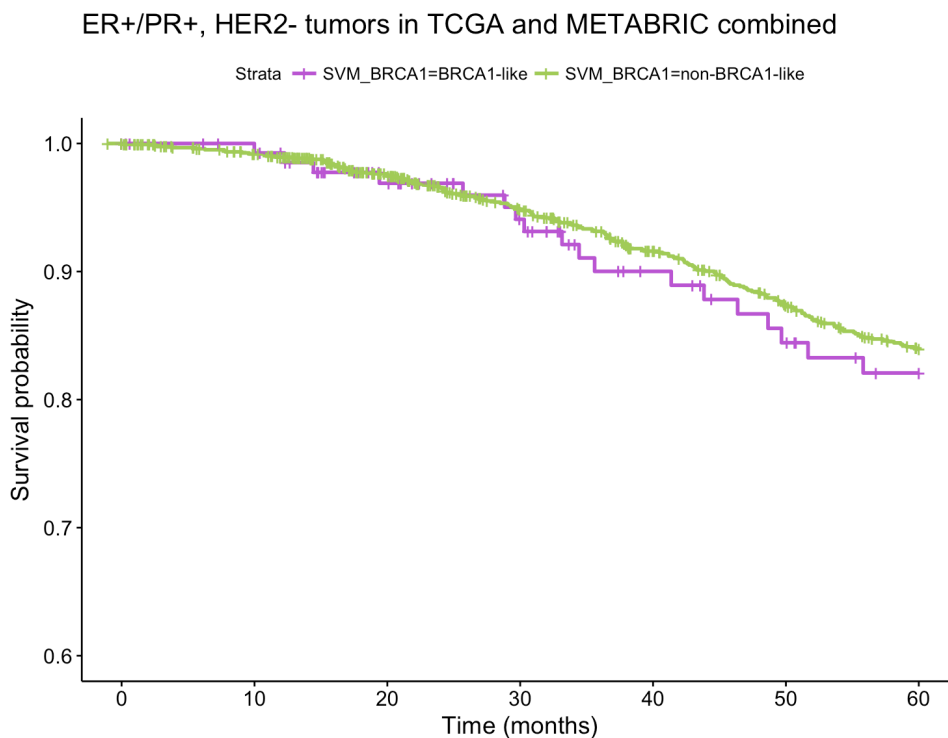
**(D)**



**Supplemental Figure 2. Comparison of Ki-67 (*MKI67*) gene expression as a surrogate marker for cellular proliferation in METABRIC hormone receptor-positive breast tumors. *P*-value indicates statistical significance from a linear model adjusting for age, tumor stage, ER, PR and HER2 positivity. \*\*\*  $P < 0.001$ .**

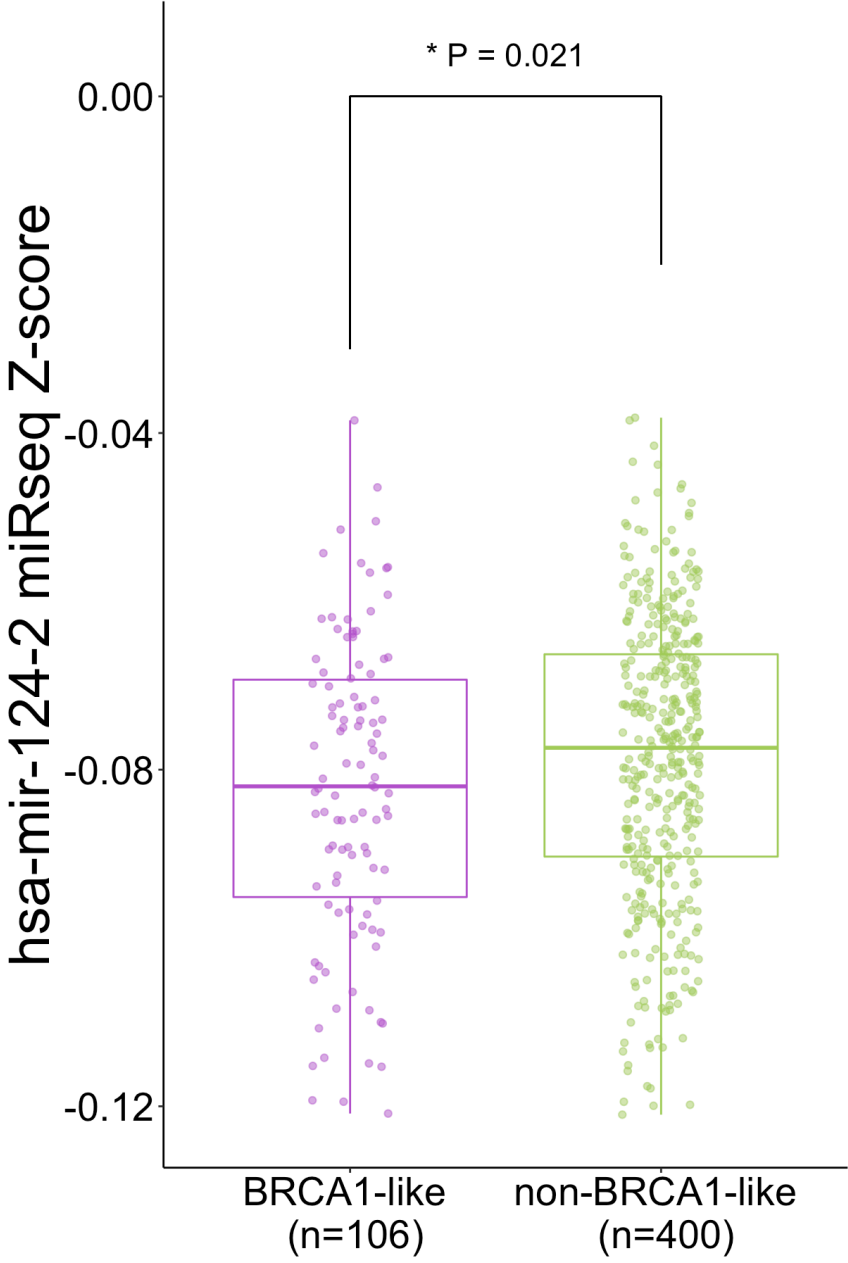


**Supplemental Figure 3. Five-year overall survival comparison between BRCA1-like and non-BRCA1-like ER-positive/PR-positive, HER2-negative breast tumors in TCGA and METABRIC (combined).** Table inset shows hazards ratio (95% CI) and *P*-value from Cox proportional hazards regression adjusting for potential confounders. \*\*\**P* < 0.001.

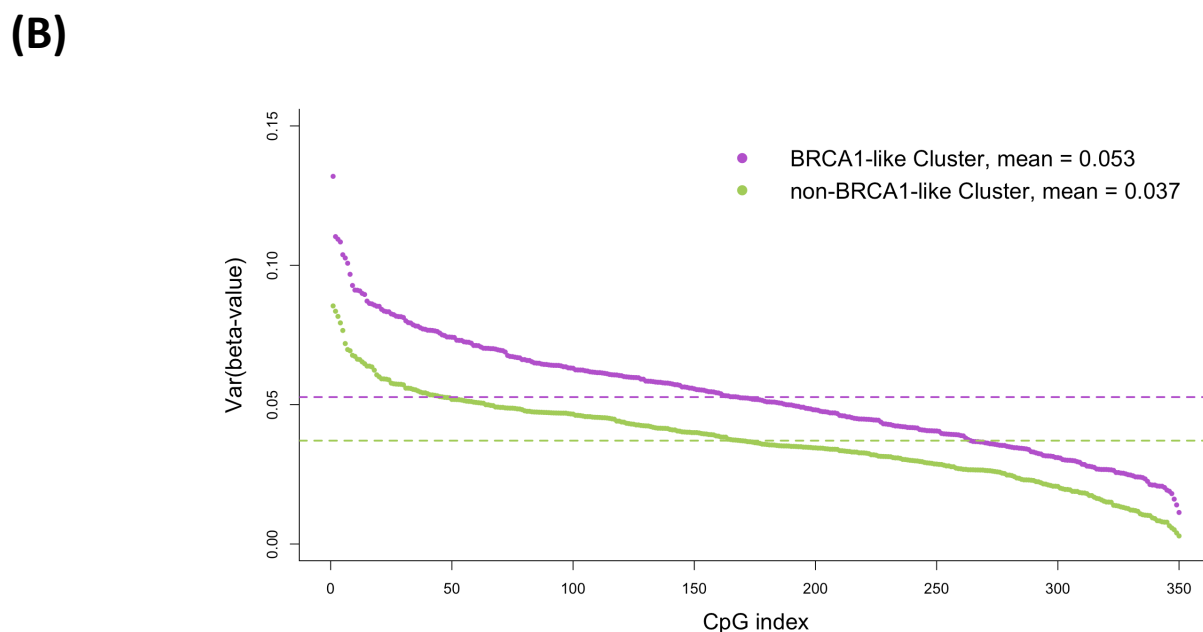
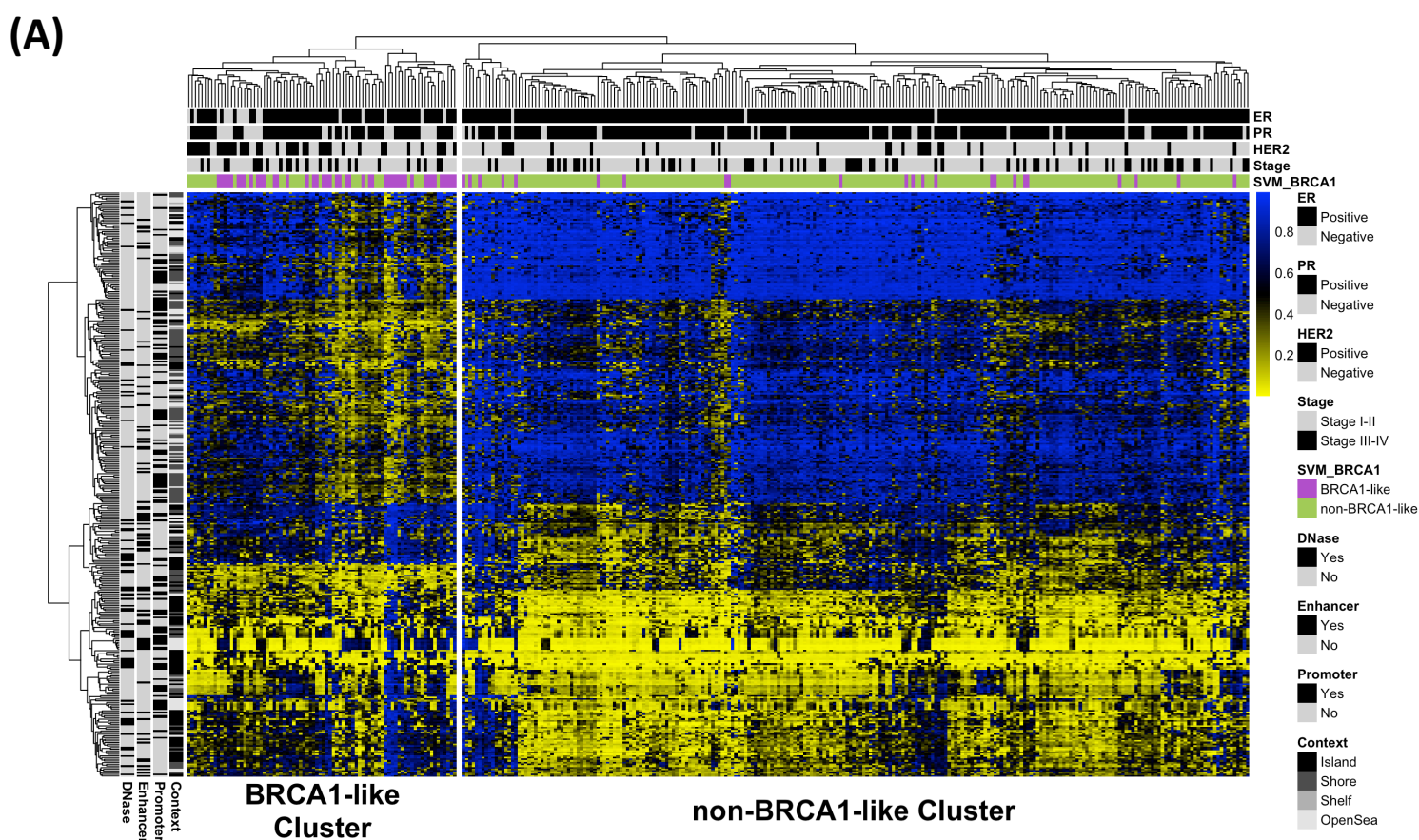


	Combined TCGA and METABRIC (ER+/PR+, HER2-)		
	n (%)	Hazards Ratio (95% CI)	<i>P</i> -value
<b>Age (years)</b>	1,391 (100.0)	1.04 (1.03-1.05)	*** 3.38E-10
<b>Stage</b>			
Stage I-II	1,225 (88.1)	1.00 (referent)	
Stage III-IV	166 (11.9)	2.74 (1.89-3.96)	*** 8.95E-08
<b>SVM BRCA1-like status</b>			
non-BRCA1-like	1,248 (89.7)	1.00 (referent)	
BRCA1-like	143 (10.3)	1.18 (0.73-1.92)	0.50

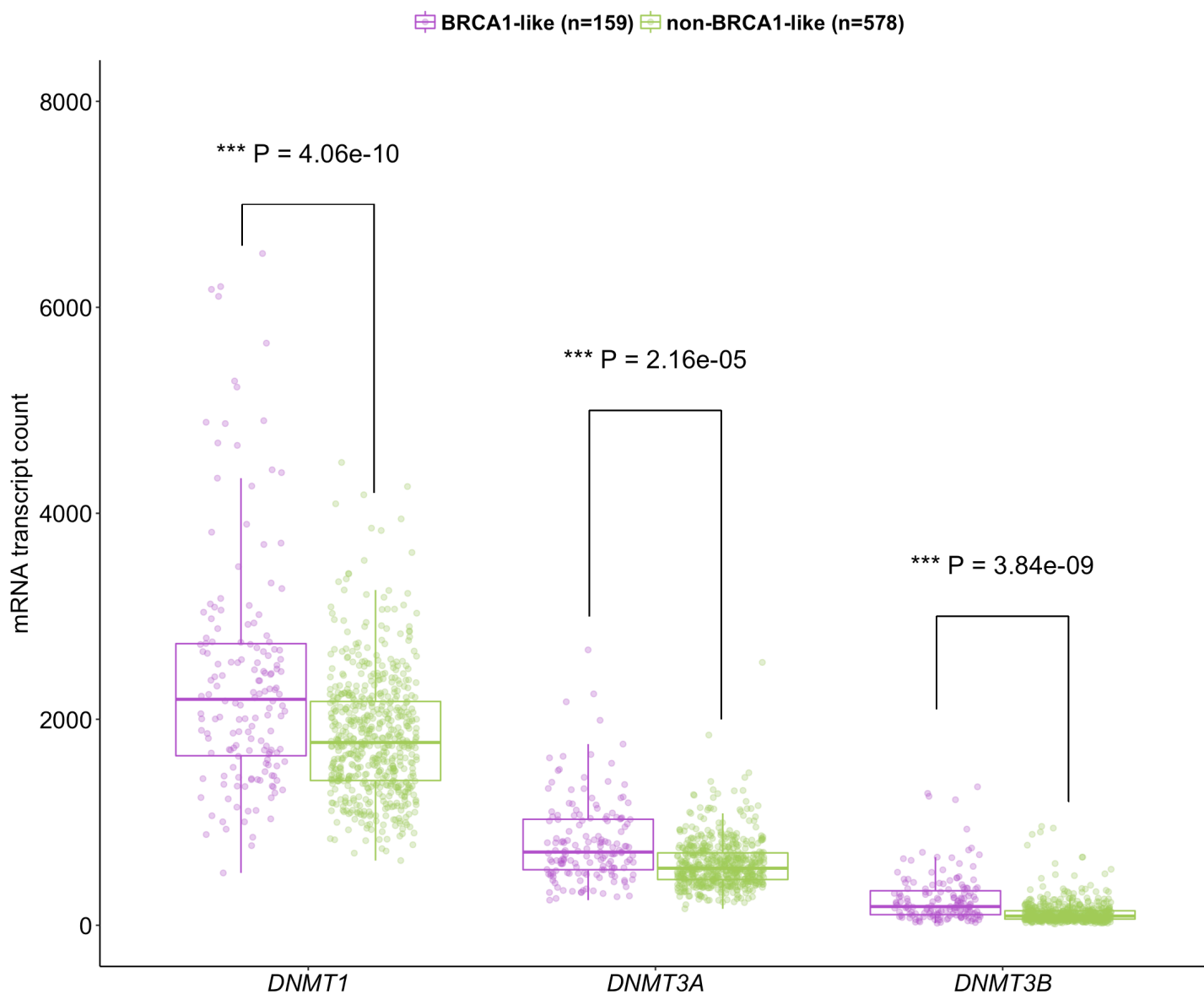
**Supplemental Figure 4. miR124-2 with hypermethylation exhibit reduced gene expression in TCGA BRCA1-like receptor positive tumors.** *P*-value indicates statistical significance from a linear model adjusting for age, tumor stage, ER, PR and HER2 positivity. \**P* < 0.05.



**Supplemental Figure 5. Comparison of heterogeneity between the “BRCA1-like methylation cluster” and “non-BRCA1-like methylation cluster” generated by hierarchical clustering of 350 most differential CpGs identified by DMRcate (all FDR < 0.05 and  $|\log_2\Delta\text{beta}| \geq 3.50$ ). (A) Heat map showing unsupervised clustering (Euclidean distance, complete linkage) of the 350 DMRcate-identified CpGs. (B) Rank-ordered inter-sample variance in beta-values of the 350 differentially methylated CpGs. Horizontal dotted lines indicate mean inter-sample variance distribution for each group.**



**Supplemental Figure 6. Differential gene expression of DNA methyltransferases (*DNMT1/3A/3B*) in TCGA receptor-positive BRCA1-like breast tumors.** A *P*-value indicates statistical significance from linear model adjusting for age, tumor stage, ER, PR and HER2 positivity. \*\*\**P* < 0.001.



**Supplemental Figure 7. Comparison of Somatic Mutational Signature 1 contributed to by genome-wide cytosine-to-thymine (C>T) deamination events in TCGA hormone receptor-positive breast tumors. A *P*-value indicates statistical significance from linear model adjusting for age, tumor stage, ER, PR and HER2 positivity. \*\**P* < 0.01.**

