	Specificity	Sensitivity	Misclassification	Correct classification
Mutation status [14]	0.74	0.37	0.39	0.61
Chromosomal instability [15]	0.61	0.54	0.41	0.59
Fibroblast serum response [16]	0.46	0.67	0.47	0.53
Wound serum response [17]	0.53	0.6	0.44	0.56
Hypoxia signature [18]	0.47	0.59	0.49	0.51
Radioresistance signature [19]	0.28	0.8	0.54	0.46
NKI Local recurrence signature [10]	0.53	0.63	0.43	0.57
Invasiveness signature [20]	0.78	0.4	0.35	0.65
Local recurrence signature [9]	0.35	0.77	0.51	0.49
70-gene NKI signature [21]	0.6	0.53	0.42	0.58
P53 signature [22]	0.37	0.72	0.51	0.49
Recurrence score [23]	0.62	0.49	0.42	0.58
Rotterdam signature [24]	0.55	0.54	0.45	0.55
PTEN-loss signature [25]	0.54	0.61	0.43	0.57
Genomic grade [26]	0.55	0.62	0.43	0.57
Proliferation signature [27]	0.46	0.72	0.45	0.55
Molecular portraits [28]	0.49	0.64	0.46	0.54
ER+ breast cancer [29]	0.53	0.62	0.44	0.56
Pooled signature [30]	0.61	0.57	0.4	0.6
Proliferation markers [31]	0.57	0.61	0.42	0.58
Three modules signature [32]	0.56	0.55	0.44	0.56
Wound signature [8]	0.53	0.6	0.44	0.56

Table S1: Accuracy of 22 signatures for predicting LR in the French samples. Training sets were generated by cross-validation (10 fold - 100 permutations), and Diagonal Linear Discriminant Analysis was applied to classify patients who had either experienced LR within the first 10 years (119) or remained free from LR for at least 10 years after primary treatment (224).

	HER2+	Luminal A	Luminal B	Luminal-HER	Triple Negative
Group 1	0	**126 (71.59) ***	30 (17.04)	17 (9.66)	3 (1.7)
Group 2	0	9 (16.36)	**42 (76.36) ***	4 (7.27)	0
Group 3	**14 (23.73) ***	6 (11.17)	18 (30.50)	**17 (28.81) ***	4 (6.78)
Group 4	0	0	2 (3.77)	0	**51 (96.23) ***

Table S2: Composition of hierarchical clustering groups. Hierarchical clustering of the 343

tumors and the 5000 most variant probes resulted in the formation of four sub-clusters, closely related to the subtype definition. The composition of the four subgroups (%) is presented.
* Significant (p<0.05) hyper-geometric tests for luminal A tumors in Group 1 (p=2.86e-36), luminal B in Group 2 (p=1.60e-18), HER2+ and luminal-HER in Group 3 (p=0 and p=3.72e-06), and for triple negative tumors in Group 4 (p=1.46e-53).

	Patients with no LR, n=224 (%)	**Patients with LR, n=119 (%)**
Luminal A	104 (73.76)	37 (26.24)
Luminal B	55 (59.78)	37 (40.22)
Luminal-HER	21 (55.26)	17 (44.74)
Triple Negative	38 (65.52)	20 (34.48)
HER2+	6 (42.86)	8 (57.14)

Table S3: Association of tumor molecular subtype assignment based on the gene expression of ESR1, PR, ERBB2 and AURKA of 343 primary breast carcinomas with LR status (χ^2 test, p = 0.033).

Bological Process – GO id	**Description**	**Pvalue**	**BC subtype**
GO:0050865	regulation of cell activation	0.000	TNBC
GO:0050868	negative regulation of T cell activation	0.000	TNBC
GO:0010942	positive regulation of cell death	0.000	TNBC
GO:0051249	regulation of lymphocyte activation	0.000	TNBC
GO:0055082	cellular chemical homeostasis	0.000	TNBC
GO:0002695	negative regulation of leukocyte activation	0.000	TNBC
GO:0050801	ion homeostasis	0.000	TNBC
GO:0030003	cellular cation homeostasis	0.000	TNBC
GO:0043067	regulation of programmed cell death	0.000	TNBC
GO:0031347	regulation of defense response	0.001	TNBC
GO:0032944	regulation of mononuclear cell proliferation	0.001	TNBC
GO:0055066	di-, tri-valent inorganic cation homeostasis	0.001	TNBC
GO:0016265	death	0.001	TNBC
GO:0032735	positive regulation of interleukin-12 production	0.001	TNBC

GO:0006082	organic acid metabolic process	0.000	HER2+
GO:0042180	cellular ketone metabolic process	0.000	HER2+

Table S4: Gene Ontology enrichment of differentially expressed genes (TNBC=Triple-Negative Breast Cancer) between tumors from patients with LR compared to those without LR.

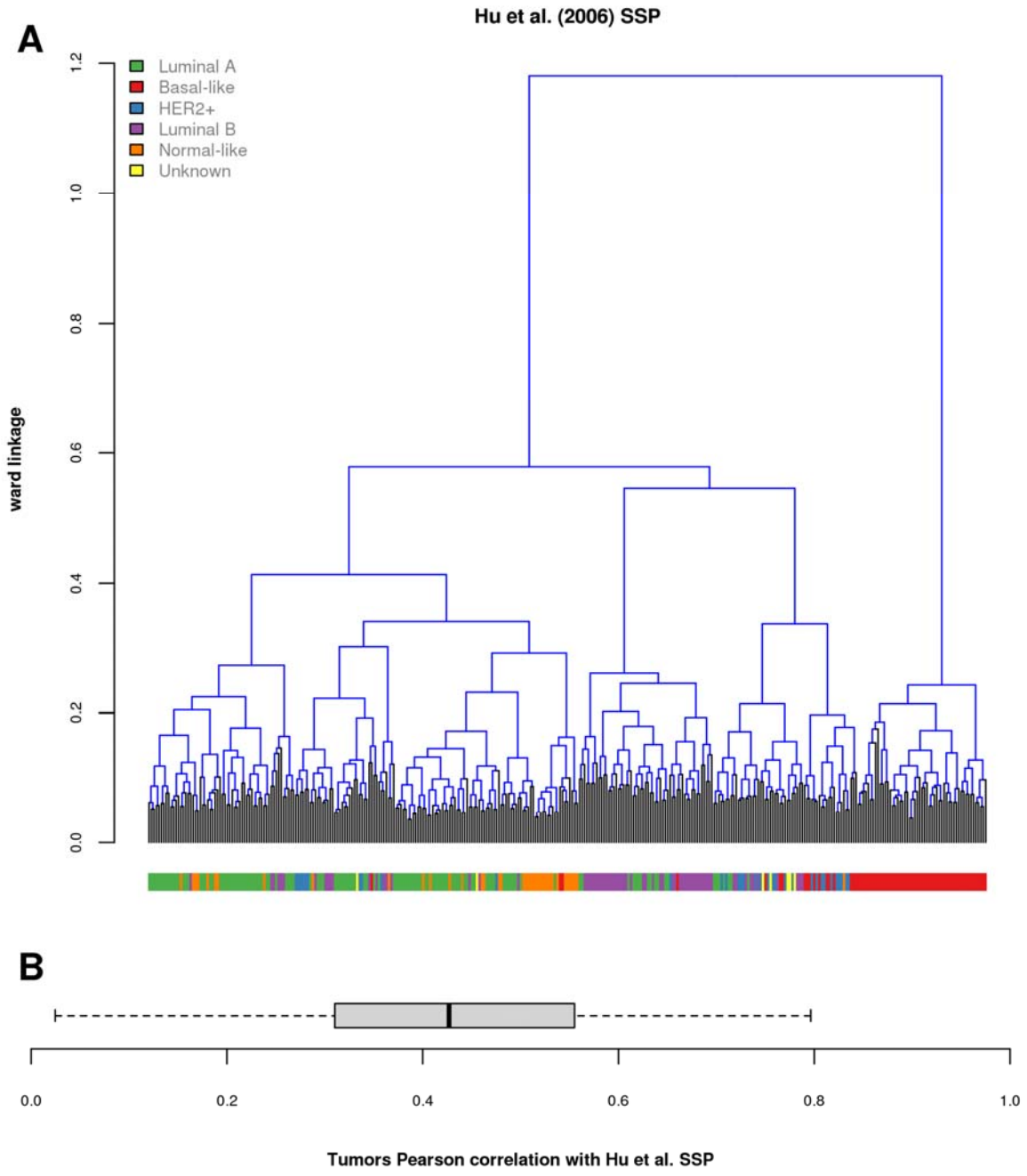

Figure S1: Class assignment using the Hu et al. SSP. A. Hierarchical clustering of the 343 tumors samples with the predicted Hu et al. subtype (luminal A, luminal B, HER2+, Basal-like,

Normal-like). **B.** Pearson correlation of the 343 gene-centered tumors with the centroids of the Hu et al. subtypes. Each sample was assigned to a subtype based on its largest correlation. The samples with largest correlation less than 0.1 were left unclassified.