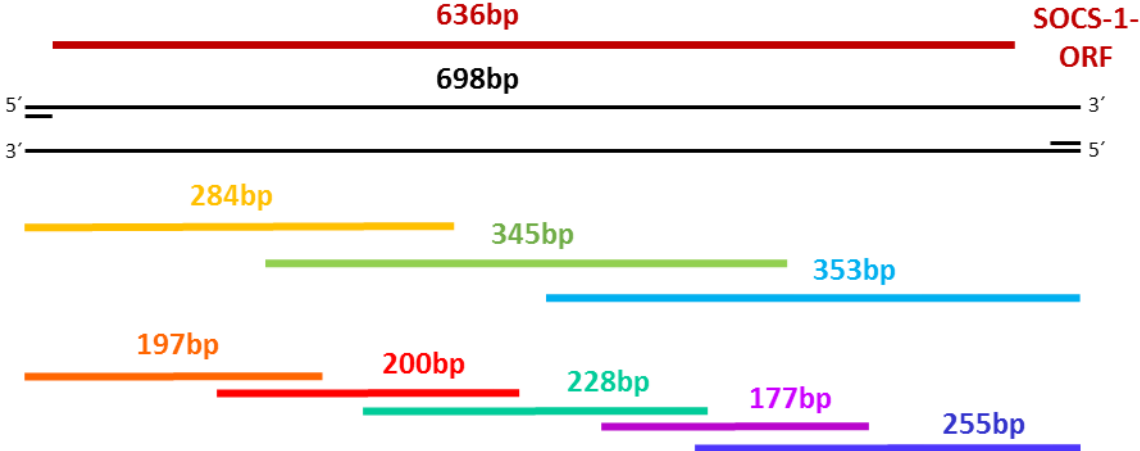
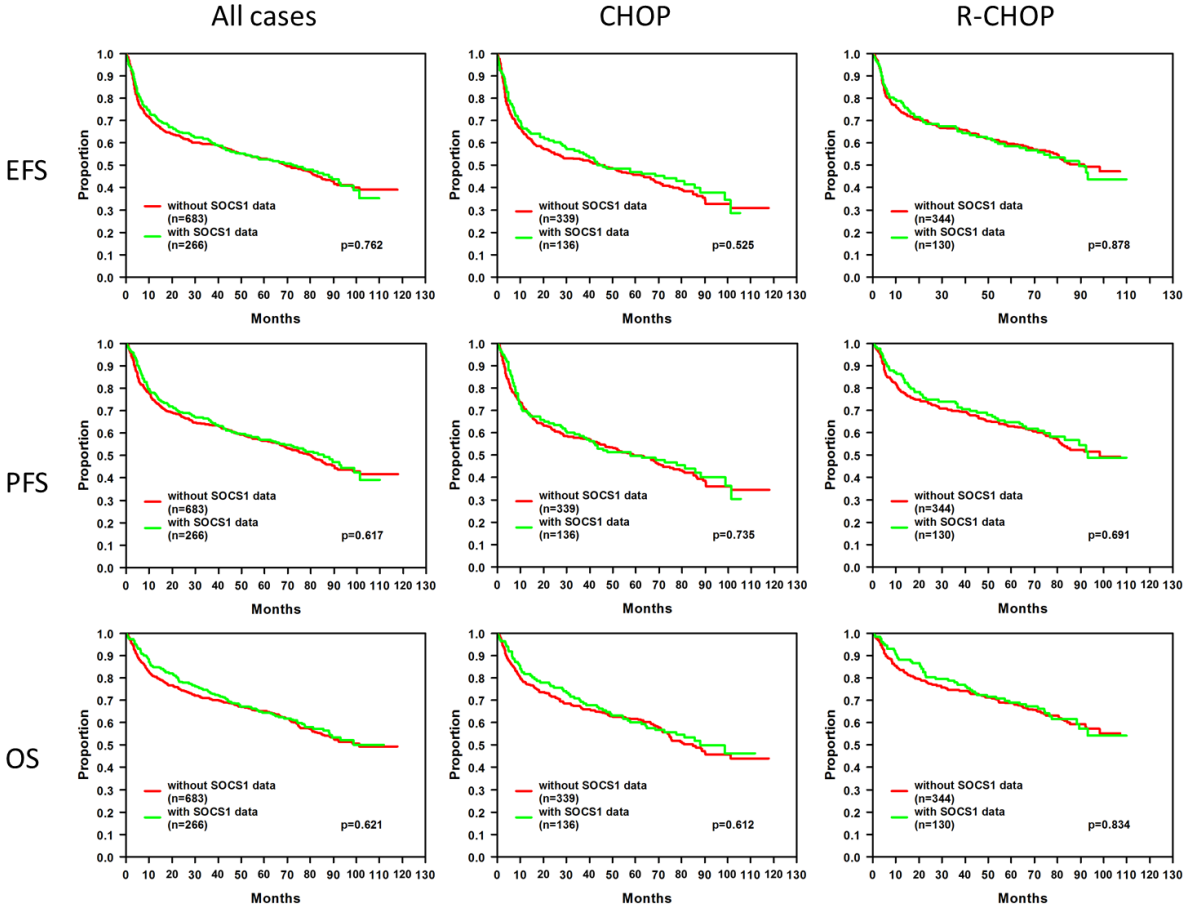


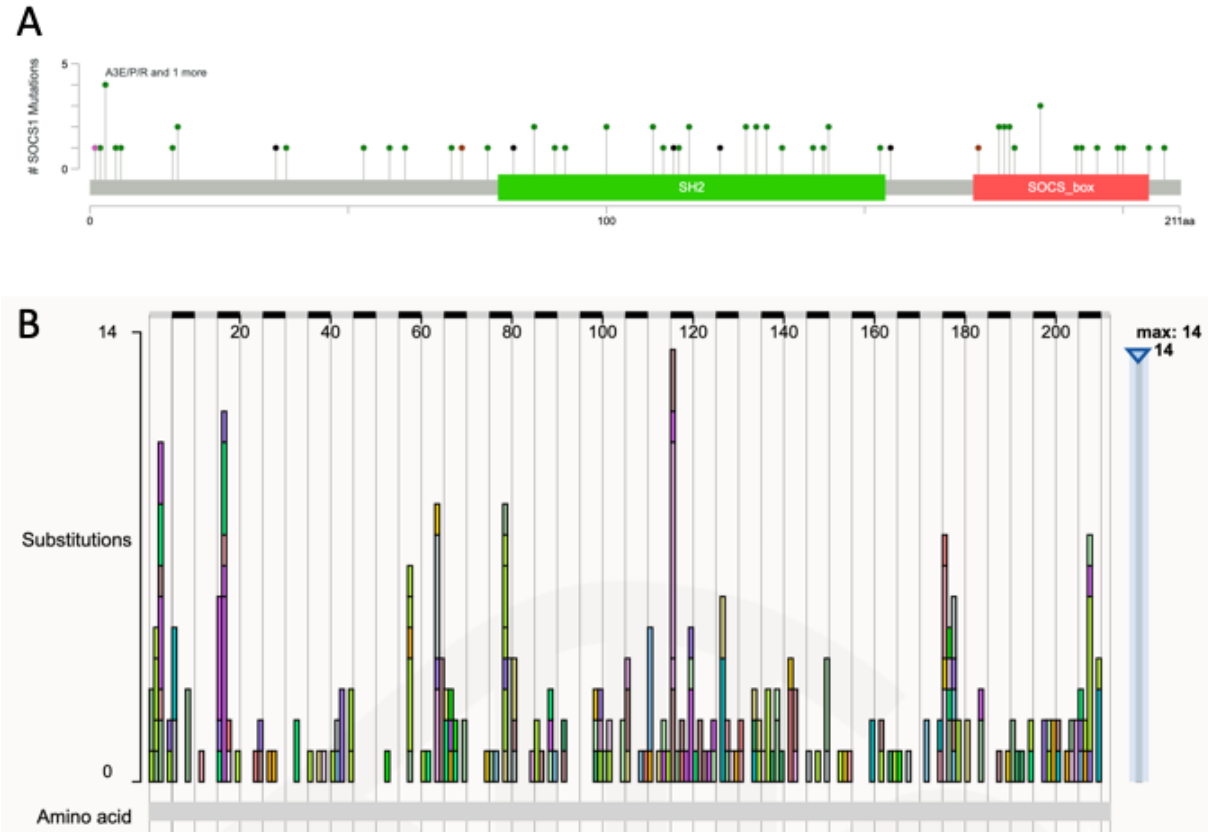
Supplementary Data



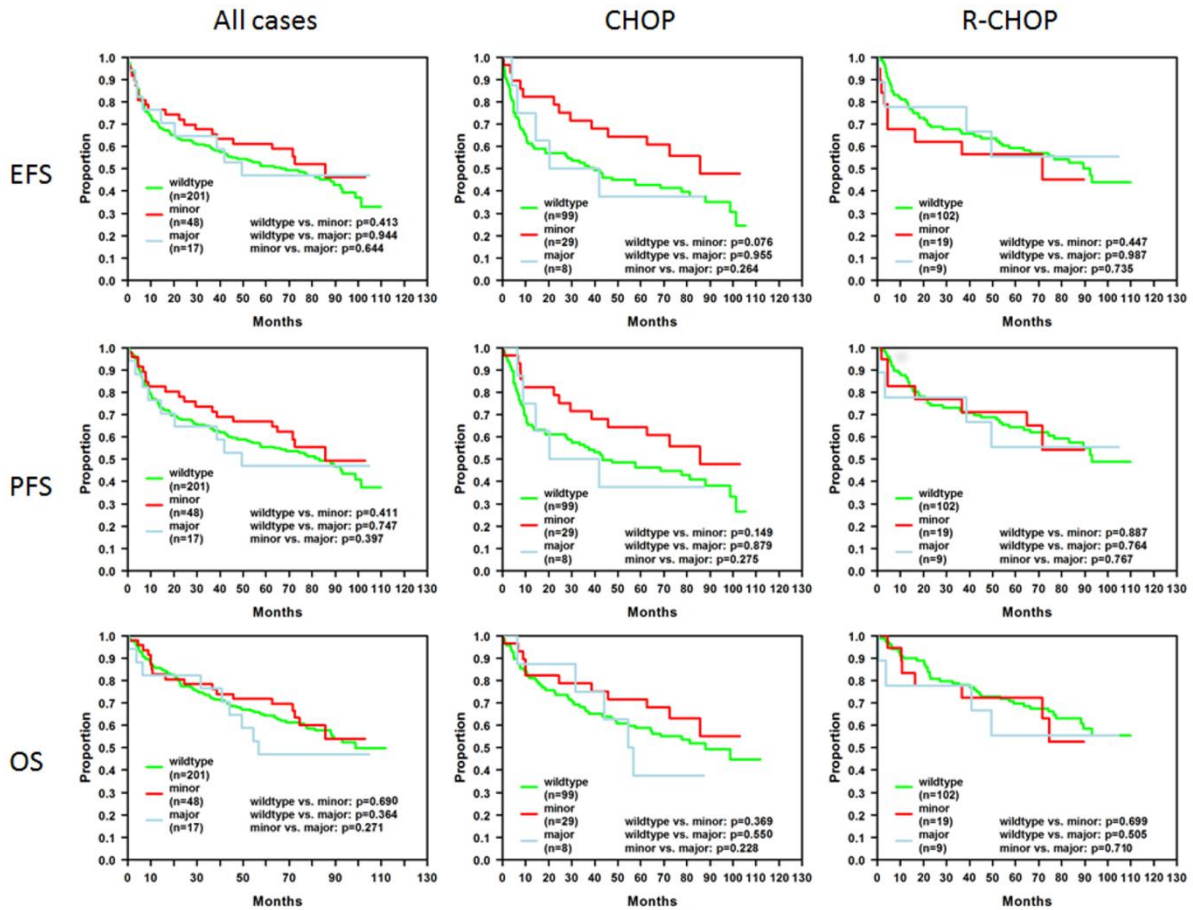
Supplementary Figure 1: Schematic position and length of amplicons used to cover the SOCS1 open reading frame.



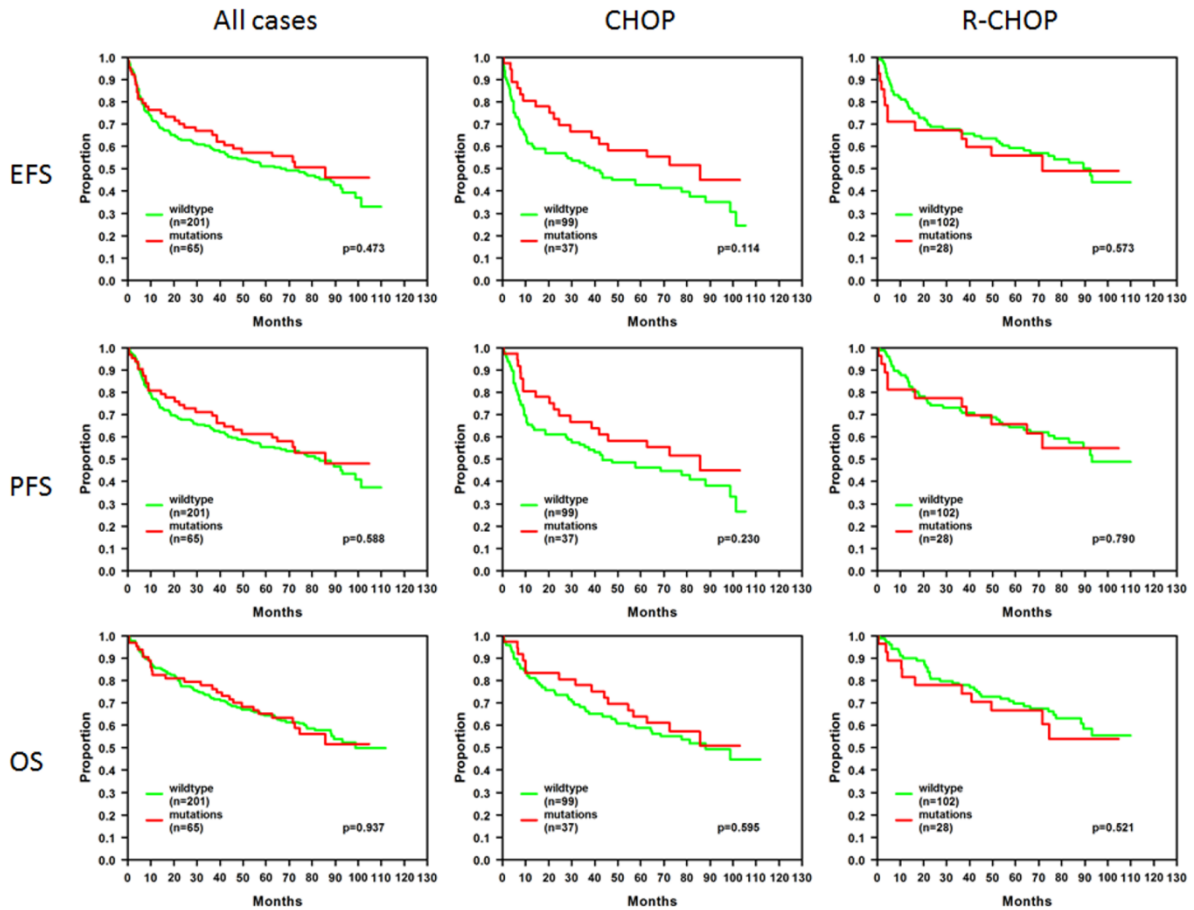
Supplementary Figure 2: The subgroup examined represents the whole RICOVER-60 cohort. The survival time analyses of the patients of the RICOVER-60 trial are shown with (green) or without (red) *SOCS1* mutation data with respect to all cases (A) or the patients' treatment (B: CHOP-treated; C: R-CHOP-treated). The analyses were conducted for event-free survival (EFS), progression-free survival (PFS), and overall survival (OS). The corresponding numbers of patients (n) and results of statistical testing for differences (p) are shown. No significant differences were detected.



Supplementary Figure 3: Comparison of positional accumulation of variants across *SOCS1* using publicly available databases across different cancer types. A. cBioportal, B. COSMIC.



Supplementary Figure 4: Survival curves of DLBCL patients related to SOCS1 mutation subtypes. The survival time analyses of the patients of the RICOVER 60 trial are shown with wildtype (green), minor mutated (red), or major mutated (light blue) SOCS1 with respect to all cases (A) or patient treatments (B: CHOP-treated; C: R-CHOP-treated). The analyses were conducted for event-free survival (EFS), progression-free survival (PFS), and overall survival (OS). The corresponding numbers of patients (n) and results of statistical testing for differences (p) are shown. No significant differences were detected.



Supplementary Figure 5: Survival of DLBCL patients related to overall SOCS1 mutations. The survival time analyses of the patients of the RICOVER 60 trial are shown with wildtype (green) or mutated (red) SOCS-1 with respect to all cases (A) or patient treatments (B: CHOP-treated; C: R-CHOP-treated). The analyses were calculated for event-free survival (EFS), progression-free survival (PFS), and overall survival (OS). The corresponding numbers of patients (n) and results of statistical testing for differences (p) are shown. No significant differences were detected

Supplementary Table 1: Primer sequences of PCR primers used to cover the *SOCS1* open reading frame. Either 3 longer (L) or 5 shorter amplicons (S) were used. Primers were named as follows: numbers indicate the amplicon counted from 3' to 5'. L or S indicates a long or a short amplicon. f= forward primer, r= reverse revers primer.

Primer	Sequence 5'-3'
1Lf = 1Sf	GGCTGGCCCCCTTCTGTAG
1Lr = 2Sr	CCCCGTGCACGCTCA
2Lf = 3Sf	GCACTTCCGCACATTCCGTT
2Lr = 4Sr	TGGCGCAGCGGGGCCCCCAGCAT
3Lf = 4Sf	GAAGTCTTTTTTCGCCCTTA
3Lr = 5Sr	ACGGCATCCCAGTTAATGCT
1Sr	AACGGAATGTGCGGAAGTGC
2Sf	TTCCTCCTCTTCCTCCT
3Sr	GAAGAGGCAGTCGAAGCTCT
5Sf	AGAGCTTCGACTGCCTCTTC

Supplementary Table 2: A: Summary of patient characteristics, immunohistochemistry and fluorescence in situ hybridization data. Grouping of the patients with respect to the *SOCS1* mutation status, three different classifiers and the treatment with or without rituximab. No significant differences were found. B: Immunohistochemical detection of characteristic markers in DLBCL and FISH analyses. Patients were grouped into patients with neutral and patients with pathogenic *SOCS1* mutations. Grouping of the patients with respect to the *SOCS1* mutation status, three different classifiers and the treatment with or without rituximab. Significant differences were detected in the expressions of HLADR independently from the specific *SOCS1* mutation classifier. No further differences could be detected.

B	wildtype (n=163)	minor (n=37)	major (n=14)	p-value	wildtype (n=163)	mutations (n=51)	p-value	putative neutral (n=184)	putative defect (n=30)	p-value
Morphology:										
Immunoblastic	15 (12%)	1 (4%)	0 (0%)	0.508	15 (12%)	1 (3%)	0.196	16 (12%)	0 (0%)	0.131
Centroblastic	113 (88%)	24 (96%)	9 (100%)		113 (88%)	33 (97%)		123 (89%)	23 (100%)	
IHC:										
BCL2 (51-100%)	102/152 (67%)	23/34 (68%)	7/14 (50%)	0.445	102/152 (67%)	30/48 (63%)	0.557	114/173 (66%)	18/27 (67%)	0.937
BCL6 (26-100%)	109/138 (79%)	28/33 (85%)	13/13 (100%)	0.153	109/138 (79%)	41/46 (89%)	0.125	124/156 (80%)	26/28 (93%)	0.093
MYC (41-100%)	32/123 (26%)	10/29 (35%)	3/10 (30%)	0.629	32/123 (26%)	13/39 (33%)	0.374	35/139 (25%)	10/23 (44%)	0.070
CD5 (1-100%)	10/145 (7%)	2/34 (6%)	1/14 (7%)	1.000	10/145 (7%)	3/48 (6%)	1.000	11/166 (7%)	2/27 (7%)	1.000
CD10 (1-100%)	51/149 (34%)	11/34 (32%)	3/14 (21%)	0.693	51/149 (34%)	14/48 (29%)	0.517	55/169 (33%)	10/28 (36%)	0.741
HLADR (1-100%)	135/151 (89%)	25/34 (74%)	9/13 (69%)	0.015	135/151 (89%)	34/47 (72%)	0.004	150/171 (88%)	19/27 (70%)	0.035
MUM1 (5-100%)	123/146 (84%)	29/34 (85%)	10/12 (83%)	1.000	123/146 (84%)	39/46 (85%)	0.930	141/167 (84%)	21/25 (84%)	1.000
Ki67 (76-100%)	101/142 (71%)	22/34 (65%)	9/12 (75%)	0.773	101/142 (71%)	31/46 (67%)	0.630	117/163 (72%)	15/25 (60%)	0.230
MYC+/BCL2+	24/106 (23%)	9/25 (36%)	1/8 (13%)	0.334	24/106 (23%)	10/33 (30%)	0.371	26/119 (22%)	8/20 (40%)	0.095
FISH:										
BCL2 positive	18/149 (12%)	7/33 (21%)	2/12 (17%)	0.276	18/149 (12%)	9/45 (20%)	0.179	21/169 (12%)	6/25 (24%)	0.127
BCL6 positive	52/152 (34%)	9/34 (27%)	4/12 (33%)	0.700	52/152 (34%)	13/46 (28%)	0.452	57/172 (33%)	8/26 (31%)	0.810
MYC positive	12/150 (8%)	2/34 (6%)	1/13 (8%)	1.000	12/150 (8%)	3/47 (6%)	1.000	13/171 (8%)	2/26 (8%)	1.000
IGH positive	37/90 (41%)	6/12 (50%)	2/8 (25%)	0.606	37/90 (41%)	8/20 (40%)	0.927	41/97 (42%)	4/13 (31%)	0.428
MYC+/BCL2+	2/116 (2%)	2/23 (9%)	0/8 (0%)	0.196	2/116 (2%)	2/31 (7%)	0.196	3/130 (2%)	1/17 (6%)	0.392
MYC+/BCL6+	1/115 (1%)	0/25 (0%)	0/8 (0%)	1.000	1/115 (1%)	0/33 (0%)	1.000	1/129 (1%)	0/19 (0%)	1.000
BCL2+/BCL6+	4/115 (4%)	1/24 (4%)	0/7 (0%)	1.000	4/115 (4%)	1/31 (3%)	1.000	4/128 (3%)	1/18 (6%)	0.487
MYC+/BCL2+/BCL6+	0/110 (0%)	0/22 (0%)	0/7 (0%)	-	0/110 (0%)	0/29 (0%)	-	0/123 (0%)	0/16 (0%)	-
Hans classifier:										
ABC	78/136 (57%)	17/32 (53%)	7/11 (64%)	0.824	78/136 (57%)	24/43 (56%)	0.859	90/154 (58%)	12/25 (48%)	0.328
GCB	58/136 (43%)	15/32 (47%)	4/11 (36%)		58/136 (43%)	19/43 (44%)		64/154 (42%)	13/25 (52%)	
Lymph 2Cx***:										
ABC	65/140 (46%)	14/34 (41%)	5/10 (50%)	0.800	65/140 (46%)	19/44 (43%)	0.731	74/158 (47%)	10/26 (39%)	0.527
GCB	59/140 (42%)	15/34 (44%)	3/10 (30%)	(global)	59/140 (42%)	18/44 (41%)	(global)	66/158 (42%)	11/26 (42%)	(global)
intermediate/unclassified	16/140 (11%)	5/34 (15%)	2/10 (20%)	0.832	16/140 (11%)	7/44 (16%)	0.909	18/158 (11%)	5/26 (19%)	0.654
				(ABC vs. GCB)			(ABC vs. GCB)			(ABC vs. GCB)
IHC-score**:										
0, 1	75/119 (63%)	14/27 (52%)	8/10 (80%)	0.287	75/119 (63%)	22/37 (60%)	0.696	84/133 (63%)	13/23 (57%)	0.545
2, 3	44/119 (37%)	13/27 (48%)	2/10 (20%)		44/119 (37%)	15/37 (41%)		49/133 (37%)	10/23 (44%)	
FISH/IHC-score**:										
0, 1	68/113 (60%)	12/24 (50%)	8/9 (89%)	0.130	68/113 (60%)	20/33 (61%)	0.965	76/127 (60%)	12/19 (63%)	0.783
2, 3, 4	45/113 (40%)	12/24 (50%)	1/9 (11%)		45/113 (40%)	13/33 (39%)		51/127 (40%)	7/19 (37%)	
P53 (26-100%)										
P53 mutated	26/146 (18%)	5/36 (14%)	0/12 (0%)	0.332	26/146 (18%)	5/48 (10%)	0.225	27/166 (16%)	4/28 (14%)	1.000
	28/109 (26%)	5/18 (28%)	2/8 (25%)	1.000	28/109 (26%)	7/26 (27%)	0.897	30/120 (25%)	5/15 (33%)	0.535

* sample according to Horn et al. Blood 2013 with SOCS1 data

** Horn et al. Blood 2013

*** sample according to Staiger et al. JCO 2017 submitted/review with SOCS1 data