SUPPLEMENTAL APPENDIX

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Methods

RNA extraction and cDNA synthesis

Total RNA extraction was performed from 10^7 cells using AllPrep Mini Kit (Qiagen, Hilden, Germany) according to the manufacturer's instructions. Reverse transcription reaction was run with 2 µg total RNA using TaqMan® Reverse transcription Kit (Applied Biosystems, Foster City, CA) and contained the following reagents: TaqMan® 10X RT Buffer (1x), $MgCl_2$ (5.5 mM), nucleoside triphosphates (2.5 mM), random hexamers (2.5 µM), RNase inhibitor (0.4 U/µl) and MultiScribe™ reverse transcriptase (1.25 U/µl). The total volume was 30 µl. Reaction

conditions were 25°C for 10 minutes (primer incubation), 48°C for 30 minutes (reverse transcription), and 95°C for 5 minutes (inactivation of reverse transcriptase).

RT-qPCR reaction

RT-qPCR reactions were carried out using the MicroAmp™ Fast Optical 96-Well Reaction Plate (Applied Biosystems, Foster City, CA). The total volume for quantification of *RUNX1-RUNX1T1* or *B2M* transcript levels was 25 µl based on the following protocol: 2,5 µl cDNA, 12,5 µl TaqMan® Universal PCR Master Mix (2x), 3 pmol/µl of forward and reverse primers, as well as 1 pmol/µl of FAM/TAMRA labeled *RUNX1-RUNX1T1* or 2 pmol/µl of *B2M* probe, respectively. PCR conditions were: 50°C for 2 minutes and 95°C for 10 minutes (denaturation), followed by 50 cycles at 95°C for 15 seconds (annealing) and 60°C for 60 seconds (extension).

RT-qPCR analyses for *RUNX1-RUNX1T1* and *B2M* were performed in triplicates and included patient samples, negative template controls (NTC) and serial plasmid dilutions (10^2-10^6) for the respective genes. For sensitivity calculation, RNA of Kasumi-1 cell line (*RUNX1-RUNX1T1*-positive) and a patient sample were serially diluted in HL-60 cells (*RUNX1-RUNX1T1*-negative). In general, negativity of NTCs, correlation coefficient of the standard curve ≥0.990, PCR efficiency of ≥85% and B2M ≥50,000 copy numbers were set as compulsory standards for each run. *RUNX1-RUNX1T1* transcript levels were reported as normalized values of *RUNX1-RUNX1T1* per 10^6 transcripts of the housekeeping gene *B2M* [*RUNX1-RUNX1T1* transcript levels/*B2M* transcript levels × 10^6]. As previously published by our group, the following equation was applied for calculation of *RUNX1-RUNX1T1* and *B2M* transcript levels: $10^{(C - Y)/S}$ [C = average cycle threshold (C_t) value from triplicates; Y = Y-intercept; S = slope of the standard curve].[1] RT-qPCR negativity was defined as C_t >Y-intercept. The maximum sensitivity of our assay was 10^{-6}. All procedures are accredited under the DIN EN ISO/IEC 17025:2005 and DIN EN ISO 15189:2014 of the Deutsche Akkreditierungsstelle GmbH.

Statistical Analyses

The definition of CR, relapse, event-free survival (EFS), relapse-free survival (RFS), and overall survival (OS) were based on recommended criteria.[2] The cumulative incidence of relapse (CIR) was calculated according to Gray.[3] Survival times were calculated from the date the MRD sample was obtained. The median follow-up for survival was calculated according to the method of Korn.[4] Logistic and Cox proportional hazards models were used to identify

prognostic variables for CR, OS, and CIR.[5,6] For *RUNX1-RUNX1T1* transcript levels, a $\log_{10}$ transformation of the data was performed (hazard ratio [HR] for 10-fold higher value). In addition, $\log_{10}$ transformation necessitated substitution of 0 by the first *RUNX1-RUNX1T1* transcript level defined as negative, 0.33. Additional covariables in multivariable analysis were age, BM blast counts, lactate dehydrogenase (LDH) serum levels, white blood cell counts (WBC) as continuous variable, and *KIT* mutation status as dichotomous variable. Determining cut-off values of prognostic factors in survival data with competing risks was done based on maximally selected log-rank statistics.[7] Comparisons of the distribution of *RUNX1-RUNX1T1* transcript levels according to qualitative group variables were performed using the Mann-Whitney U test. Correlations between continuous variables were calculated using the Spearman rank test. Survival distributions were calculated using the Kaplan-Meier method, and differences between two groups were analyzed using the two-sided log-rank test. Cumulative incidence functions were compared using Gray's test. An effect was considered significant if P<5%. All statistical analyses were performed with the statistical software environment R version 3.2.1, using the R packages rms version 4.1-1, survival version 2.41-3, cmprsk version 2.2-7, and IBM SPSS Statistics 25.

References

1. Corbacioglu A, Scholl C, Schlenk RF, et al. Prognostic impact of minimal residual disease in CBFB-MYH11-positive acute myeloid leukemia. *J Clin Oncol.* 2010;28(23):3724-3729.
2. Döhner H, Estey EH, Amadori S, et al. Diagnosis and management of acute myeloid leukemia in adults: recommendations from an international expert panel, on behalf of the European LeukemiaNet. *Blood.* 2010;115(3):453-474.
3. Gray R. A class of *K*-sample tests for comparing the cumulative incidence of a competing risk. *Ann Stat.* 1988;16(3):1141-1154.
4. Korn EL. Censoring distributions as a measure of follow-up in survival analysis. *Stat Med.* 1986;5(3):255-260.
5. Therneau TM, Grambsch PM. *Modeling Survival Data: Extending the Cox Model,* Springer (New York); 2000.
6. Cox DR. Regression models and life-tables. *J R Stat Soc B.* 1972;34(2):187-220.
7. Woo SY, Kim S, Kim J. Determining cutoff values of prognostic factors in survival data with competing risks. *CompStat*. 2016;31(1): 369–386.

Supplemental Table T1. Distribution of BM and PB samples during therapy and follow-up.

Number of samples:

Time point	BM samples, n	PB samples, n
Diagnosis	**145**	**135**
Cycle 1	**111**	**91**
Cycle 2	**110[§]**	**101[#]**
Cycle 3	**105**	**87**
Cycle 4	**99**	**95**
Cycle 5	**73**	**62**
Cycle 6	**8**	**7**
End of treatment (EOT)	**99**	**86**
3 months (after EOT)	**78**	**72**
6 months (after EOT)	**61**	**53**
9 months (after EOT)	**47**	**44**
12 months (after EOT)	**46**	**46**
15 months (after EOT)	**36**	**39**
18 months (after EOT)	**41**	**46**
21 months (after EOT)	**31**	**33**
24 months (after EOT)	**23**	**26**
27 months (after EOT)	**14**	**17**
30 months (after EOT)	**18**	**19**
33 months (after EOT)	**7**	**9**
36 months (after EOT)	**11**	**18**
39 months (after EOT)	**5**	**5**
42 months (after EOT)	**6**	**9**
45 months (after EOT)	**4**	**6**
48 months (after EOT)	**4**	**9**

Abbreviations: EOT, end of treatment; BM, bone marrow; PB, peripheral blood. [§]60 patients received induction II, 50 patients received consolidation I; [#]58 patients received induction II, 43 patients received consolidation I.

Supplemental Table T2: Baseline characteristics and outcome according to trial cohorts.

Study	HD98A[#] (n=14)	AMLSG 07-04[#] (n=43)	AMLSG 11-08[§] (n=32)	AMLSG 21-13[§] (n=45)	Other (n=21)	*P* value
Median age, years (range)	42 (19 – 59)	38 (18 – 57)	52 (27 – 73)	50 (21 – 76)	56 (19 – 72)	0.0002
Male sex, n (%)	9 (64)	23 (53)	18 (56)	26 (58)	10 (48)	0.8882
AML history, n (%)						
De novo	14 (100)	41 (95)	27 (84)	33 (89)	17 (85)	0.5118
Secondary	0 (0)	0 (0)	1 (3)	1 (3)	0 (0)	
Therapy-related	0 (0)	2 (5)	4 (13)	3 (8)	3 (15)	
Missing data, n	0	0	0	8	1	
Median WBC, x 10^9/l (range)	9.3 (1.0 – 60.5)	10.0 (1.2 – 48.2)	9.0 (1.4 – 45.7)	7.8 (1.6 – 118)	7.7 (1.9 – 45)	0.8269
Missing data, n	0	1	0	7	1	
Median platelet count, x 10^9/l (range)	37 (16 – 226)	23 (5 – 303)	32 (8 – 279)	34 (5 – 246)	32 (3 – 119)	0.7152
Missing data, n	0	1	0	7	1	
Median hemoglobin, g/dl (range)	8.7 (4.2 – 14.8)	8.8 (4.6 – 15.1)	9.2 (5.2 – 13.2)	8.9 (3.8 – 12.5)	9.0 (4.8 – 11.2)	0.2194
Missing data, n	0	1	0	7	1	
Median peripheral blood blasts, % (range)	52 (16 – 90)	47 (4 – 99)	35 (0 – 85)	39 (0 – 82)	40 (0 – 79)	0.3820
Missing data, n	1	4	1	11	1	
Median bone marrow blasts, % (range)	45 (6 – 85)	65 (20 – 100)	60 (13 – 95)	50 (15 – 100)	58 (17 – 91)	0.2412
Missing data, n	3	0	1	8	2	
Median LDH, U/l (range)	530 (213 – 1,662)	476 (138 – 3,550)	484 (226 – 1,968)	428 (5 – 1,910)	417 (205 – 4,420)	0.6610
Missing data, n	9	1	0	7	0	
***KIT* mutation, n (%)**	4 (29)	17 (41)	6 (19)	7 (21)	5 (28)	0.2204
Missing data, n	0	2	1	11	3	
***FLT3*-ITD/TKD mutation, n (%)**	1 (7)	3 (8)	3 (9)	4 (9)	3 (14)	0.9348
Missing data, n	0	4	0	0	0	
***NRAS* mutation, n (%)**	2 (14)	8 (20)	6 (19)	6 (17)	2 (11)	0.9435
Missing data, n	0	2	0	10	3	
***ASXL2* mutation, n (%)**	6 (46)	7 (17)	2 (7)	0 (0)	3 (23)	0.0240
Missing data, n	1	1	3	45	8	
Response						
CR after cycle I	9 (64)	35 (81)	26 (81)	35 (78)	14 (67)	0.5046
RD after cycle I	1 (7)	2 (5)	0 (0)	0 (0)	0 (0)	
PR after cycle I	4 (29)	6 (14)	6 (19)	10 (22)	7 (33)	

CR after cycle II	14 (100)	43 (100)	32 (100)	42 (98)	21 (100)	0.9544
RD after cycle II	0 (0)	0 (0)	0 (0)	1 (2)	0 (0)	
Outcome						
OS						0.2992
Median, years	4.95	NR	NR	NR	NR	
3-year survival rate (%)	57	84	71	78	77	
4-year survival rate (%)	57	80	71	NR	77	
EFS						0.6568
Median, years	1.52	7.95	NR	NR	NR	
3-year survival rate (%)	50	67	68	70	58	
4-year survival rate (%)	43	67	68	NR	58	
RFS						0.5165
Median, years	1.27	7.87	NR	NR	NR	
3-year survival rate (%)	50	70	68	69	54	
4-year survival rate (%)	43	70	68	NR	54	

[#] included patients 18 to 60 years; [§] included patients ≥18 years (no age limit)
NR, not reached

Supplemental Table T3: Multivariable analysis for response to induction I.

	CR after cycle 1	
	OR (95% CI)	***P***
	n=120	
Log_{10}TL diagnosis BM	0.93 (0.32-2.72)	0.895
***KIT*^{mut}**	0.28 (0.11-0.76)	0.012
Age	1.00 (0.97-1.04)	0.936
BM blasts	1.01 (0.99-1.03)	0.258
LDH	1.00 (1.00-1.00)	0.055
WBC	0.98 (0.95-1.02)	0.984

Supplemental Table T4: Multivariable analysis determining the prognostic significance of $MR^{3.0}$ after cycle 2 in BM and PB.

	Relapse	
	HR (95% CI)	***P***
Transcript level reduction after cycle 2	**n=92**	
BM		
$MR^{3.0}$	0.48 (0.24-0.98)	0.043
***KIT*^{mut}**	2.03 (0.97-4.22)	0.059
Age	1.00 (0.97-1.03)	0.959
BM blasts	1.00 (0.98-1.01)	0.773
LDH	1.00 (1.00-1.00)	0.983
WBC	1.00 (0.98-1.03)	0.971
PB	**n=81**	
$MR^{3.0}$	0.35 (0.14-0.85)	0.021
***KIT*^{mut}**	1.72 (0.77-3.83)	0.184
Age	1.00 (0.97-1.03)	0.875
BM blasts	1.01 (0.99-1.02)	0.626
LDH	1.00 (1.00-1.00)	0.525
WBC	1.01 (0.99-1.03)	0.262

Supplemental Table T5: Multivariable analyses stratified according to dasatinib treatment determining the prognostic significance of *RUNX1-RUNX1T1* transcript levels, MRD cut-off values, and MRD^{neg} at EOT in BM and PB.

	Death		**Relapse**	
	HR (95% CI)	***P***	**HR (95% CI)**	***P***
Transcript Level	**n=81**		**n=82**	
BM				
Log_{10}TL	1.71 (1.12-2.62)	0.013	1.76 (1.21-2.54)	0.003
***KIT*^{mut}**	2.87 (0.97-8.48)	0.056	2.08 (0.84-5.13)	0.114
Age	1.02 (0.98-1.07)	0.362	1.00 (0.97-1.04)	0.909
BM blasts	0.99 (0.97-1.01)	0.368	1.00 (0.98-1.02)	0.977
LDH	1.00 (1.00-1.00)	0.938	1.00 (1.00-1.00)	0.769
WBC	1.03 (1.00-1.05)	0.064	1.01 (0.97-1.04)	0.754
PB	**n=71**		**n=72**	
Log_{10} TL	1.61 (1.06-2.43)	0.025	2.91 (1.79-4.74)	<0.001
***KIT*^{mut}**	4.33 (1.41-13.31)	0.011	3.07 (1.24-7.60)	0.015
Age	1.01 (0.97-1.06)	0.595	1.00 (0.97-1.04)	0.811
BM blasts	0.99 (0.97-1.02)	0.564	1.01 (0.99-1.03)	0.522
LDH	1.00 (1.00-1.00)	0.898	1.00 (1.00-1.00)	0.570
WBC	1.02 (0.99-1.05)	0.220	1.00 (0.97-1.04)	0.813
Specific cut-off values	**n=81**		**n=82**	
BM				
Absolute TL >83	4.88 (1.47-16.26)	0.010	3.98 (1.60-9.89)	0.003
***KIT*^{mut}**	2.62 (0.86-8.02)	0.091	2.07 (0.84-5.09)	0.112
Age	1.03 (0.98-1.08)	0.207	1.01 (0.98-1.05)	0.506
BM blasts	0.99 (0.97-1.01)	0.382	1.00 (0.98-1.02)	0.991
LDH	1.00 (1.00-1.00)	0.828	1.00 (1.00-1.00)	0.747
WBC	1.02 (1.00-1.05)	0.076	1.01 (0.98-1.04)	0.754
PB	**n=71**		**n=72**	
Absolute TL >5	3.36 (1.06-10.66)	0.040	5.40 (2.01-14.51)	0.001
***KIT*^{mut}**	3.92 (1.29-11.88)	0.016	3.12 (1.27-7.66)	0.013
Age	1.01 (0.97-1.05)	0.750	1.00 (0.97-1.03)	0.961
BM blasts	1.00 (0.97-1.02)	0.756	1.00 (0.98-1.02)	0.682
LDH	1.00 (1.00-1.00)	0.701	1.00 (1.00-1.00)	0.473
WBC	1.02 (0.99-1.05)	0.181	1.01 (0.98-1.04)	0.679
MRD^{neg}	**n=81**		**n=82**	
BM				
MRD^{neg}	0.22 (0.05-0.88)	0.033	0.39 (0.15-0.97)	0.042
***KIT*^{mut}**	2.48 (0.84-7.34)	0.100	2.13 (0.88-5.15)	0.094
Age	1.01 (0.97-1.06)	0.565	1.00 (0.97-1.04)	0.924
BM blasts	0.99 (0.97-1.02)	0.503	1.00 (0.98-1.02)	0.836
LDH	1.00 (1.00-1.00)	0.986	1.00 (1.00-1.00)	0.782
WBC	1.03 (1.00-1.05)	0.076	1.00 (0.97-1.04)	0.864
PB	**n=71**		**n=72**	
MRD^{neg}	0.28 (0.09-0.81)	0.019	0.37 (0.16-0.87)	0.022
***KIT*^{mut}**	3.44 (1.12-10.54)	0.031	2.24 (0.95-5.30)	0.067
Age	1.01 (0.97-1.05)	0.628	1.01 (0.98-1.04)	0.576
BM blasts	0.99 (0.97-1.02)	0.596	1.00 (0.98-1.02)	0.999
LDH	1.00 (1.00-1.00)	0.813	1.00 (1.00-1.00)	0.789
WBC	1.02 (0.99-1.05)	0.189	1.00 (0.97-1.03)	0.848

Abbreviations: MRD^{neg}, MRD negativity; EOT, end of treatment; BM, bone marrow; PB, peripheral blood; HR, hazard ratio; CI, confidence interval; *P*, p-value; n, number of patients included in the analysis; TL, transcript levels; *KIT*^{mut}, mutated *KIT* gene; LDH, lactate dehydrogenase; WBC, white blood cell count

Supplemental Table T6: Impact of additional dasatinib treatment on MRD^{neg} at EOT.

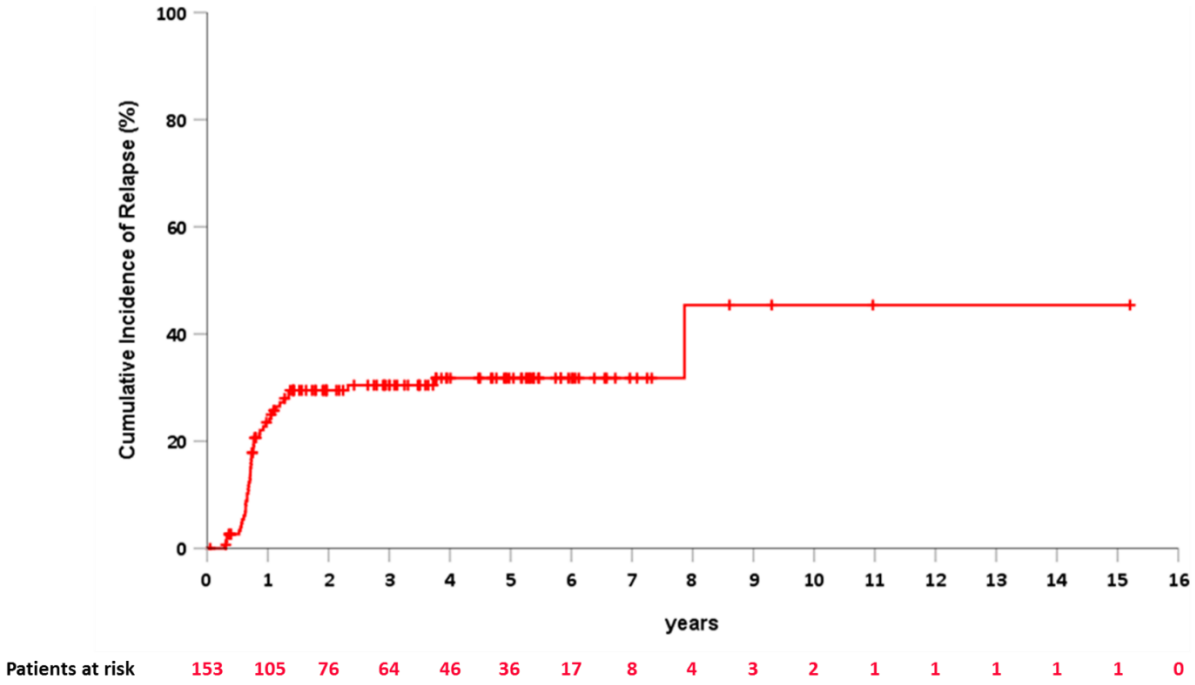
	Dasatinib (pt no)	No Dasatinib (pt no)	*P* value
EOT BM (n=99)			
MRD^{neg} EOT BM	24	27	0.0608
MRD^{pos} EOT BM	13	35	
EOT PB (n=86)			
MRD^{neg} EOT PB	28	36	0.0113
MRD^{pos} EOT PB	3	19	

Supplemental Table T7: Impact of additional dasatinib treatment on outcome.

	OS		CIR	
	HR (95% CI)	***P***	**HR (95% CI)**	***P***
Dasatinib treatment				
BM				
After Cycle 1	0.95 (0.45-1.99)	0.881	0.49 (0.20-1.08)	0.077
After Cycle 2	0.82 (0.36-1.89)	0.644	0.54 (0.24-1.19)	0.124
At EOT	0.89 (0.33-2.40)	0.814	0.75 (0.32-1.75)	0.509
PB				
After Cycle 1	1.19 (0.48-2.96)	0.710	0.68 (0.30-1.57)	0.369
After Cycle 2	1.21 (0.48-3.02)	0.691	0.87 (0.40-1.88)	0.718
At EOT	0.49 (0.16-1.51)	0.216	0.67 (0.29-1.54)	0.345

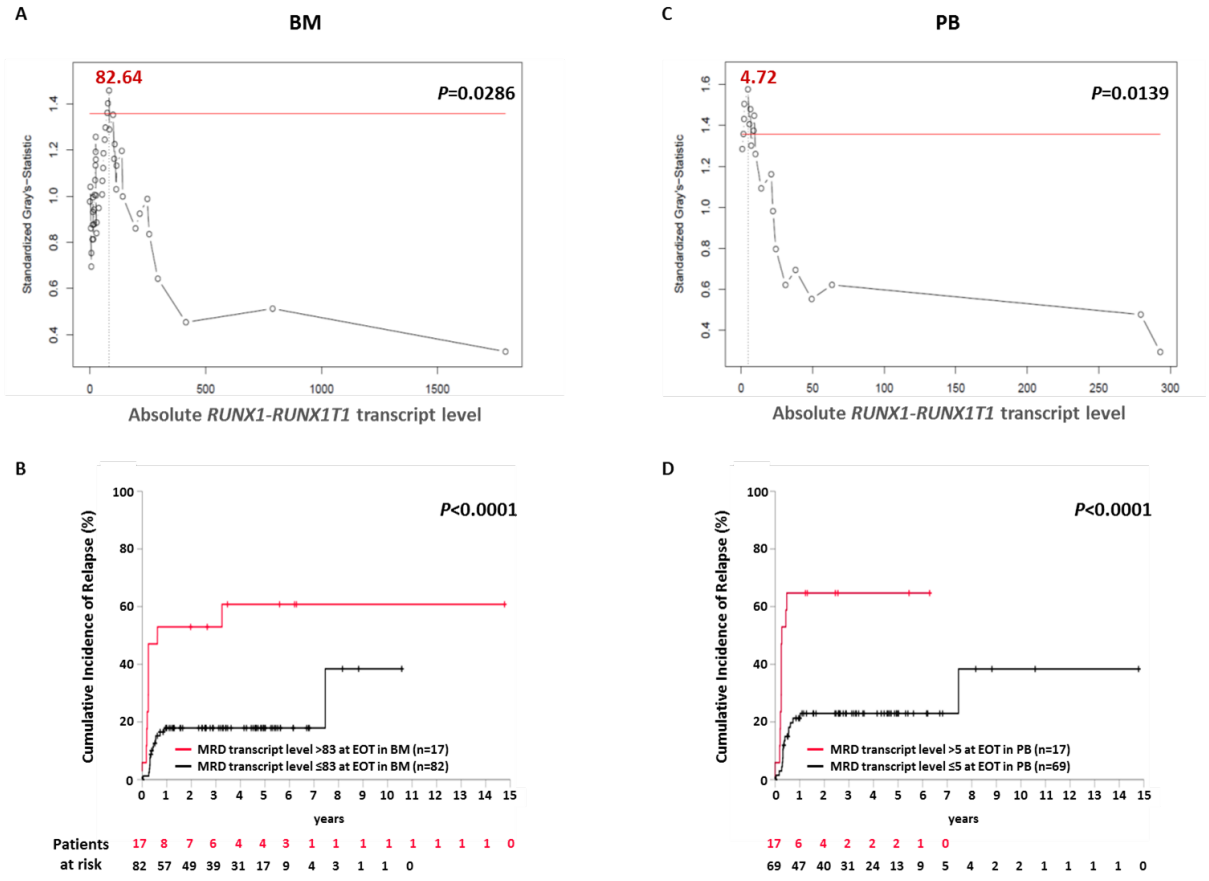
Supplemental Figure F1

Cumulative incidence of relapse (CIR) of the 155 *RUNX1-RUNX1T1*-positive AML patients.



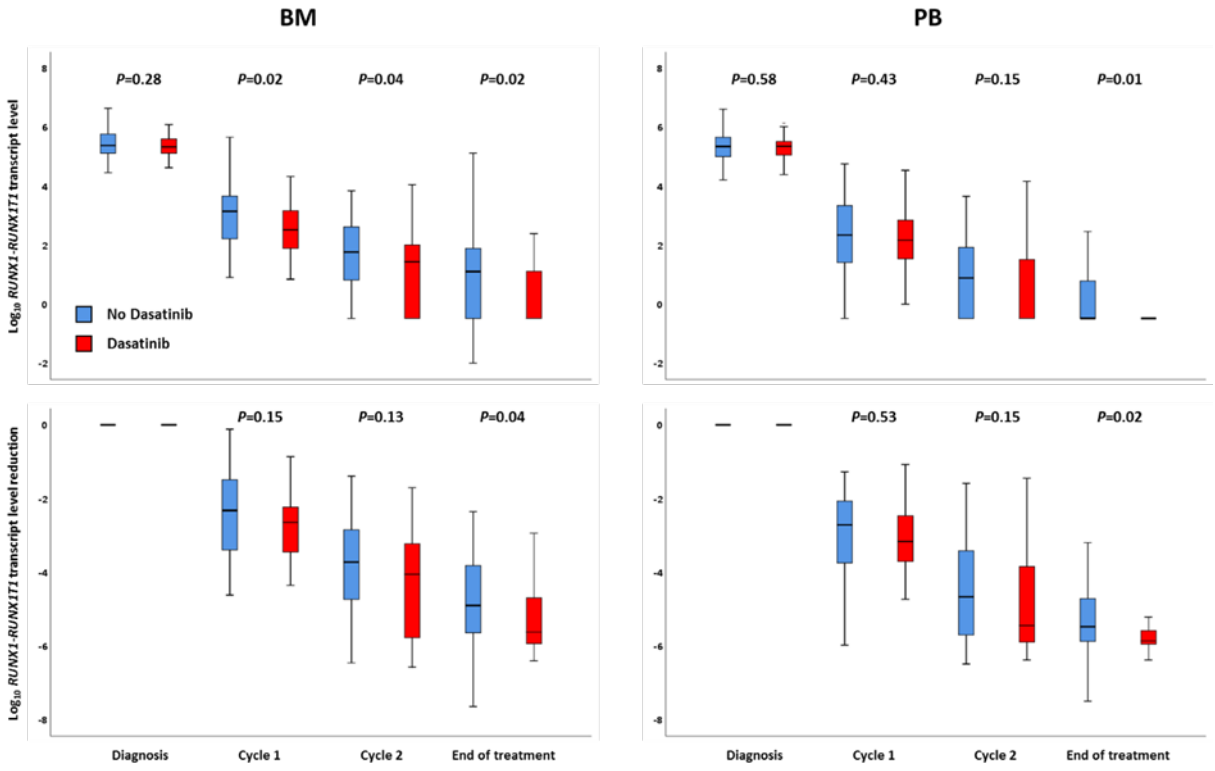
Supplemental Figure F2:

Maximally selected Gray´s statistic for competing risks delineated cut-offs at EOT being associated with a low risk of relapse. For BM, MRD level below 83 *RUNX1-RUNX1T1* transcript level predicted for superior 4-year rates of CIR (18% vs 61%; *P*<0.0001) (A and B). For PB, MRD level below 5 *RUNX1-RUNX1T1* transcript level predicted for superior 4-year rates of CIR (23% vs 65%; *P*<0.0001) (C and D).



Supplemental Figure F3:

Impact of additional dasatinib treatment on MRD kinetics. Log_{10} *RUNX1-RUNX1T1* transcript levels (upper panel) and the reduction of *RUNX1-RUNX1T1* transcript levels (lower panel) are shown for BM (left) and PB (right) during the course of treatment.



Supplemental Figure F4:

Cumulative incidence of relapse (CIR) during follow-up based on defined MRD cut-offs in BM and PB according to additional dasatinib treatment (B and D).