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Supplement Table 1. Distribution of additional cytogenetic abnormalities in Ph⁺ cells at diagnosis.

Classification	Type	Frequency, n (%)	
		Training dataset	Validation dataset
<i>ACAs in Ph⁺ cells</i>		68 (3%)	118 (3%)
High-risk ACAs in Ph ⁺ cells*	Complex aberrant karyotypes	14 (0.7%)	16 (0.5%)
	a second Ph-chromosome (+Ph)	9 (0.5%)	13 (0.4%)
	+8	7 (0.4%)	8 (0.2%)
	11q23	3 (0.2%)	5 (0.1%)
	+19	3 (0.2%)	3 (<0.1%)
	i(17q)	2 (0.1%)	3 (<0.1%)
	-7/7q-	2 (0.1%)	2 (<0.1%)
	3q26.2	1 (<0.1%)	1 (<0.1%)
Non high-risk ACAs in Ph ⁺ cells	-Y	6 (0.3%)	8 (0.2%)
	+12	2 (0.1%)	4 (0.1%)
	inv(9)	2 (0.1%)	3 (<0.1%)
	Others	17 (0.9%)	52 (2%)
<i>No ACAs in Ph⁺ cells</i>		1,887 (97%)	3,336 (97%)

ACAs, Additional chromosomal aberrations; Ph⁺ cells, Philadelphia chromosome-positive cells.

* High-risk ACAs were defined according to the 2020 ELN recommendation.

Supplement Table 2. Subjects co-variables before and after propensity-score matching analysis.

Co-variables	<i>Before propensity-score matching</i>			<i>After propensity-score matching</i>		
	Imatinib cohort (n = 1,539)	2G-TKI cohort (n = 416)	p-value	Imatinib cohort (n = 694)	2G-TKI cohort (n = 347)	p-value
Age, years, median (IQR)	41 (31, 52)	36 (28, 46)	< 0.001	36 (26, 47)	37 (25, 48)	0.47
Male, n(%)	946 (62)	249 (60)	0.55	411 (59)	202 (58)	0.76
Spleen size, cm below costal margin, median (range)	2 (0, 8)	4 (0, 13)	< 0.001	5 (0, 12)	5 (0, 12)	0.35
Sokal risk, n(%)			< 0.001			0.75
Low	689 (45)	130 (31)		259 (37)	128 (37)	
Intermediate	451 (29)	104 (25)		206 (30)	97 (28)	
High	271 (17)	123 (30)		229 (33)	122 (35)	
Unknown	128 (9)	59 (14)		-	-	
ELTS risk, n(%)			< 0.001			0.99
Low	913 (59)	202 (49)		403 (58)	202 (58)	
Intermediate	375 (24)	96 (23)		191 (28)	95 (27)	

High	123 (8)	59 (14)		100 (14)	50 (15)	
Unknown	128 (9)	59 (14)		-	-	
WBC, ×10E+9/L, median (IQR)	108 (44, 218)	163 (58, 277)	< 0.001	150 (61, 276)	153 (60, 270)	0.26
Hemoglobin, ×10E+9/L, median (IQR)	117 (98, 133)	111 (94, 129)	< 0.001	110 (92, 127)	109 (92, 126)	0.31
Platelets, ×10E+9/L, median (IQR)	403 (263, 622)	454 (289, 707)	< 0.001	444 (299, 743)	440 (296, 740)	0.69
Blood blasts, %, median (IQR)	1 (0, 2)	1 (0, 4)	< 0.001	1 (0, 4)	1 (0, 4)	0.72
Blood basophils, %, median (IQR)	5 (2, 8)	5 (3, 9)	0.01	5 (2, 8)	5 (3, 9)	0.60
Blood eosinophils, %, median (IQR)	2 (1, 4)	2 (0, 4)	0.12	2 (1, 4)	2 (1, 4)	0.85
Ph ⁺ ACAs, n (%)	51 (3)	17 (4)	0.45	28 (4)	15 (4)	0.83
High-risk ACAs, n (%)	29 (2)	12 (3)	0.21	14 (2)	10 (3)	0.38
Co-morbidity, n (%)	562 (37)	138 (33)	0.21	243 (35)	120 (35)	0.89

ACAs, Additional chromosomal aberrations; ELTS, European Treatment and Outcome Study (EUTOs) long-term survival; WBC, White blood cell.

Supplement Table 3. Uni-variable analyses on TKI-therapy failure in the training dataset.

Uni-variable Cox analyses			
Co-variate	Regression coefficient	HR (95% CI)	p-value
Male (Categorical)	0.1545	1.2 (1.0 - 1.4)	0.08
Age, years (Continuous)	0.0027	1.0 (1.0 - 1.0)	0.19
Spleen size below costal margin, cm (Continuous)	0.0782	1.1 (1.1 - 1.1)	< 0.001
WBC, ×10E+9/L (Continuous)	0.0028	1.0 (1.0 - 1.0)	< 0.001
Haemoglobin, g/L (Continuous)	-0.0238	0.9 (0.9 - 0.9)	< 0.001
Platelet, ×10E+9/L (Continuous)	0.00004	1.0 (1.0 - 1.0)	0.73
Blood blasts, % (Continuous)	0.1454	1.2 (1.1 - 1.2)	< 0.001
Blood basophils, % (Continuous)	0.0096	1.0 (1.0 - 1.0)	0.15
Blood eosinophils, % (Continuous)	0.0354	1.0 (1.0 - 1.1)	0.04
Ph ⁺ ACAs* (Categorical)			0.006
<i>No ACAs (as reference)</i>	-	-	-
<i>Non-high-risk ACAs</i>	0.4073	1.5 (0.7 - 3.0)	0.25
<i>High-risk ACAs</i>	0.7113	2.0 (1.3 - 3.2)	0.002
Co-morbidity(ies)* (Categorical)	-0.1020	0.9 (0.7 - 1.1)	0.29
Initial 2G-TKI therapy* (Categorical)	0.0446	1.0 (0.8 - 1.3)	0.69
Uni-variable Fine-Gray analyses			
Co-variate	Regression coefficient	HR (95% CI)	p-value
Male (Categorical)	0.1440	1.2 (1.0 – 1.4)	0.12
Age, years (Continuous)	0.0032	1.0 (1.0 – 1.0)	0.19
Spleen size below costal margin, cm (Continuous)	0.0777	1.1 (1.1 – 1.1)	< 0.001
WBC, ×10E+9/L (Continuous)	0.0028	1.0 (1.0 – 1.0)	< 0.001
Hemoglobin, g/L (Continuous)	-0.0237	0.9 (0.9 – 0.9)	< 0.001

Platelet, ×10E+9/L (Continuous)	0.00004	1.0 (1.0 - 1.0)	0.75
Blood blasts, % (Continuous)	0.1460	1.2 (1.1 – 1.2)	< 0.001
Blood basophils, % (Continuous)	0.0099	1.0 (1.0 – 1.0)	0.13
Blood eosinophils, % (Continuous)	0.0343	1.0 (1.0 – 1.1)	0.04
Ph ⁺ ACAs* (Categorical)			0.006
<i>No ACAs (as reference)</i>	-	-	-
<i>Non-high-risk ACAs</i>	0.4030	1.5 (0.7 - 3.1)	0.28
<i>High-risk ACAs</i>	0.7080	2.0 (1.3 – 3.2)	0.002
Co-morbidity(ies)* (Categorical)	-0.1080	0.9 (0.7 – 1.1)	0.25
Initial 2G-TKI therapy* (Categorical)	0.0407	1.0 (0.8 - 1.3)	0.71

* Ph⁺ ACAs, High-risk ACAs, comorbidity(ies) and initial 2G-TKI therapy were scored as categorical co-variables (“Y/N”) in the regression models.

2G-TKI, the second generation tyrosine kinase inhibitor; ACAs, additional chromosomal aberrations; CI, confidential interval; HR, hazard ratio.

Supplement Table 4. Multi-variable analyses in the 1,884 subjects with complete data for all the 12 candidate co-variates in the training dataset.

Multi-variable Cox analyses			
Co-variates	Regression coefficient	HR (95% CI)	p-value
Male	0.2912	1.3 (1.1, 1.6)	0.004
Age, years	0.0187	1.0 (1.0, 1.0)	< 0.001
Hemoglobin, g/L	-0.0182	0.9 (0.9, 0.9)	< 0.001
Blood blasts, %	0.1016	1.1 (1.1, 1.1)	< 0.001
Spleen size, cm below costal margin	0.0521	1.1 (1.0, 1.1)	< 0.001
High-risk ACAs in Ph ⁺ cells*	0.6049	1.8 (1.1, 3.0)	0.02
Multi-variable Fine-Gray analyses			
Co-variates	Regression coefficient	HR (95% CI)	p-value
Male	0.2821	1.3 (1.1, 1.6)	< 0.001
Age, years	0.0186	1.0 (1.0, 1.0)	< 0.001
Hemoglobin, g/L	-0.0182	0.9 (0.9, 0.9)	< 0.001
Blood blasts, %	0.0954	1.1 (1.1, 1.1)	< 0.001
Spleen size, cm below costal margin	0.0500	1.1 (1.0, 1.1)	< 0.001
High-risk ACAs in Ph ⁺ cells*	0.5793	1.8 (1.1, 2.8)	0.01

* High-risk ACAs in Ph⁺ cells was scored as categorical co-variates ("Y/N") in the regression models.

ACAs, additional chromosomal aberrations; CI, confidential interval; HR, hazard ratio.

Supplement Table 5. Subjects co-variables in the low-risk cohort by the predictive model before and after propensity-score matching analysis.

Co-variables	<i>Before propensity-score matching</i>			<i>After propensity-score matching</i>		
	Imatinib cohort (n = 1,389)	2G-TKI cohort (n = 506)	<i>p</i> -value	Imatinib cohort (n = 920)	2G-TKI cohort (n = 460)	<i>p</i> -value
Age, years, median (IQR)	37 (29, 47)	35 (28, 44)	0.001	36 (28, 47)	36 (29, 47)	0.90
Male, n(%)	706 (51)	271 (54)	0.29	548 (60)	275 (60)	0.94
Spleen size, cm below costal margin, median (range)	0 (0, 1)	0 (0, 1)	0.69	0 (0, 1)	0 (0, 1)	0.94
Sokal risk*, n(%)			0.25			0.65
Low	1,175 (85)	422 (83)		773 (84)	386 (84)	
Intermediate	153 (11)	52 (10)		110 (12)	51 (11)	
High	59 (4)	32 (7)		37 (4)	23 (5)	
Unknown	2 (<0.1)	0 (0)		0 (0)	0 (0)	
ELTS risk*, n(%)			0.81			0.78
Low	1,367 (98)	497 (98)		902 (98)	452 (98)	

Intermediate	19 (2)	9 (2)		18 (2)	8 (2)	
High	1 (<0.1)	0 (0)		0 (0)	0 (0)	
Unknown	2 (<0.1)	0 (0)		0 (0)	0 (0)	
WBC, ×10E+9/L, median (IQR)	60 (30, 120)	62 (29, 136)	0.13	56 (30, 105)	55 (26, 107)	0.92
Hemoglobin, ×10E+9/L, median (IQR)	125 (113, 139)	127 (112, 139)	0.52	132 (121, 144)	133 (123, 144)	0.72
Platelets, ×10E+9/L, median (IQR)	426 (279, 653)	437 (282, 709)	0.18	426 (276, 659)	413 (271, 695)	0.66
Blood blasts, %, median (IQR)	0 (0, 1)	0 (0, 1)	0.03	0 (0, 1)	0 (0, 1)	0.96
Blood basophils, %, median (IQR)	4 (2, 6)	4 (2, 6)	0.90	4 (2, 6)	4 (2, 6)	0.78
Blood eosinophils, %, median (IQR)	2 (0, 3)	1 (0, 2)	0.12	0 (0, 1)	0 (0, 1)	0.55
Ph ⁺ ACAs, n (%)	18 (1)	8 (2)	0.64	10 (1)	4 (1)	0.79
High-risk ACAs*, n (%)	2 (0.1)	0 (0)	1.00	1 (0.1)	0 (0)	1.00
Co-morbidity, n (%)	316 (23)	112 (22)	0.78	202 (22)	103 (22)	0.85

ACAs, Additional chromosomal aberrations; ELTS, European Treatment and Outcome Study (EUTOs) long-term survival; WBC, White blood cell.

* Fisher test.

Supplement Table 6. Subjects co-variables in the intermediate-risk cohort by the predictive model before and after propensity-score matching analysis.

Co-variables	<i>Before propensity-score matching</i>			<i>After propensity-score matching</i>		
	Imatinib cohort (n = 1,907)	2G-TKI cohort (n = 670)	<i>p</i> -value	Imatinib cohort (n = 844)	2G-TKI cohort (n = 422)	<i>p</i> -value
Age, years, median (IQR)	47 (35, 58)	42 (32, 53)	< 0.001	40 (31, 51)	40 (30, 50)	0.86
Male, n(%)	1,238 (65)	431 (64)	0.78	515 (61)	249 (59)	0.49
Spleen size, cm below costal margin, median (range)	5 (2, 9)	6 (3, 10)	< 0.001	5 (2, 10)	5 (2, 10)	0.98
Sokal risk*, n(%)			0.87			0.92
Low	541 (28)	191 (29)		245 (29)	122 (29)	
Intermediate	1,050 (55)	362 (54)		456 (54)	232 (55)	
High	315 (17)	117 (17)		143 (17)	68 (16)	
Unknown	1 (<0.1)	0 (0)		0 (0)	0 (0)	
ELTS risk*, n(%)			0.87			0.29
Low	1,059 (56)	376 (56)		456 (54)	236 (56)	

Intermediate	764 (40)	269 (40)		346 (41)	173 (41)	
High	83 (4)	25 (4)		42 (5)	13 (3)	
Unknown	1 (<0.1)	0 (0)		0 (0)	0 (0)	
WBC, ×10E+9/L, median (IQR)	154 (75, 258)	181 (100, 279)	< 0.001	189 (102, 288)	182 (101, 287)	0.50
Hemoglobin, ×10E+9/L, median (IQR)	107 (92, 122)	103 (89, 118)	0.004	101 (89, 113)	102 (90, 114)	0.91
Platelets, ×10E+9/L, median (IQR)	406 (258, 610)	401 (260, 635)	0.93	428 (274, 656)	403 (261, 621)	0.42
Blood blasts, %, median (IQR)	1 (0, 2)	1 (0, 2)	0.99	1 (0, 2)	1 (0, 3)	0.31
Blood basophils, %, median (IQR)	4 (2, 7)	4 (2, 7)	0.13	5 (2, 8)	5 (2, 8)	0.71
Blood eosinophils, %, median (IQR)	2 (1, 4)	3 (1, 5)	0.39	0 (0, 1)	0 (0, 1)	0.67
Ph ⁺ ACAs, n (%)	70 (4)	33 (5)	0.16	31 (4)	20 (3)	0.36
High-risk ACAs, n (%)	31 (2)	17 (3)	0.13	15 (2)	12 (2)	0.22
Co-morbidity, n (%)	522 (27)	184 (28)	0.96	218 (26)	111 (26)	0.86

ACAs, Additional chromosomal aberrations; ELTS, European Treatment and Outcome Study (EUTOs) long-term survival; WBC, White blood cell.

* Fisher test.

Supplement Table 7. Subjects co-variables in the high-risk cohort by the predictive model before and after propensity-score matching analysis.

Co-variables	<i>Before propensity-score matching</i>			<i>After propensity-score matching</i>		
	Imatinib cohort (n = 325)	2G-TKI cohort (n = 183)	<i>p</i> -value	Imatinib cohort (n = 158)	2G-TKI cohort (n = 158)	<i>p</i> -value
Age, years, median (IQR)	42 (32, 54)	44 (30, 53)	0.52	45 (33, 56)	47 (33, 54)	0.99
Male, n(%)	214 (66)	130 (71)	0.23	106 (67)	109 (69)	0.72
Spleen size, cm below costal margin, median (range)	16 (8, 18)	15 (9, 18)	0.78	15 (6, 17)	15 (8, 18)	0.96
Sokal risk*, n(%)			0.55			0.88
Low	6 (2)	5 (3)		5 (3)	3 (2)	
Intermediate	51 (16)	35 (19)		27 (17)	28 (18)	
High	267 (82)	142 (77)		126 (80)	127 (80)	
Unknown	1 (0.3)	1 (<1)		0 (0)	0 (0)	
ELTS risk*, n(%)			0.25			0.34
Low	11 (3)	6 (3)		3 (2)	0 (0)	

Intermediate	135 (42)	61 (33)		57 (36)	58 (37)	
High	178 (55)	115 (63)		98 (62)	100 (63)	
Unknown	1 (0.3)	1 (<1)		0 (0)	0 (0)	
WBC, ×10E+9/L, median (IQR)	246 (153, 361)	253 (160, 367)	0.69	238 (135, 353)	242 (142, 359)	0.91
Hemoglobin, ×10E+9/L, median (IQR)	86 (74, 100)	89 (77, 104)	0.32	87 (77, 98)	88 (76, 99)	0.99
Platelets, ×10E+9/L, median (IQR)	391 (266, 585)	364 (230, 608)	0.04	430 (284, 654)	395 (260, 675)	0.96
Blood blasts, %, median (IQR)	4 (1, 7)	4 (2, 8)	0.11	4 (2, 8)	4 (2, 9)	0.61
Blood basophils, %, median (IQR)	5 (2, 8)	6 (3, 9)	0.23	6 (3, 9)	6 (3, 9)	0.87
Blood eosinophils, %, median (IQR)	4 (1, 6)	4 (2, 6)	0.12	4 (1, 6)	4 (1, 6)	0.77
Ph ⁺ ACAs, n (%)	28 (9)	15 (8)	0.87	14 (9)	12 (8)	0.68
High-risk ACAs, n (%)	19 (6)	13 (7)	0.58	10 (6)	8 (5)	0.63
Co-morbidity, n (%)	69 (21)	58 (32)	0.009	40 (25)	41 (26)	0.90

ACAs, Additional chromosomal aberrations; ELTS, European Treatment and Outcome Study (EUTOs) long-term survival; WBC, White blood cell.

* Fisher test.

Supplement information

Supplement figure legends

Supplement Figure 1. Comparison outcomes between imatinib and 2G-TKI cohorts in the training dataset before and after propensity-score matching (PSM) analyses.

Supplement Figure 2. Comparison outcomes between training and validation datasets.

Supplement Figure 3. Kernel density plot of optimal cutoffs for the predictive model.

Supplement Figure 4. Performance of the predictive model for the cumulative incidence of therapy-failure.

Supplement Figure 5. Cumulative incidences of therapy-failure by different age subgroups.

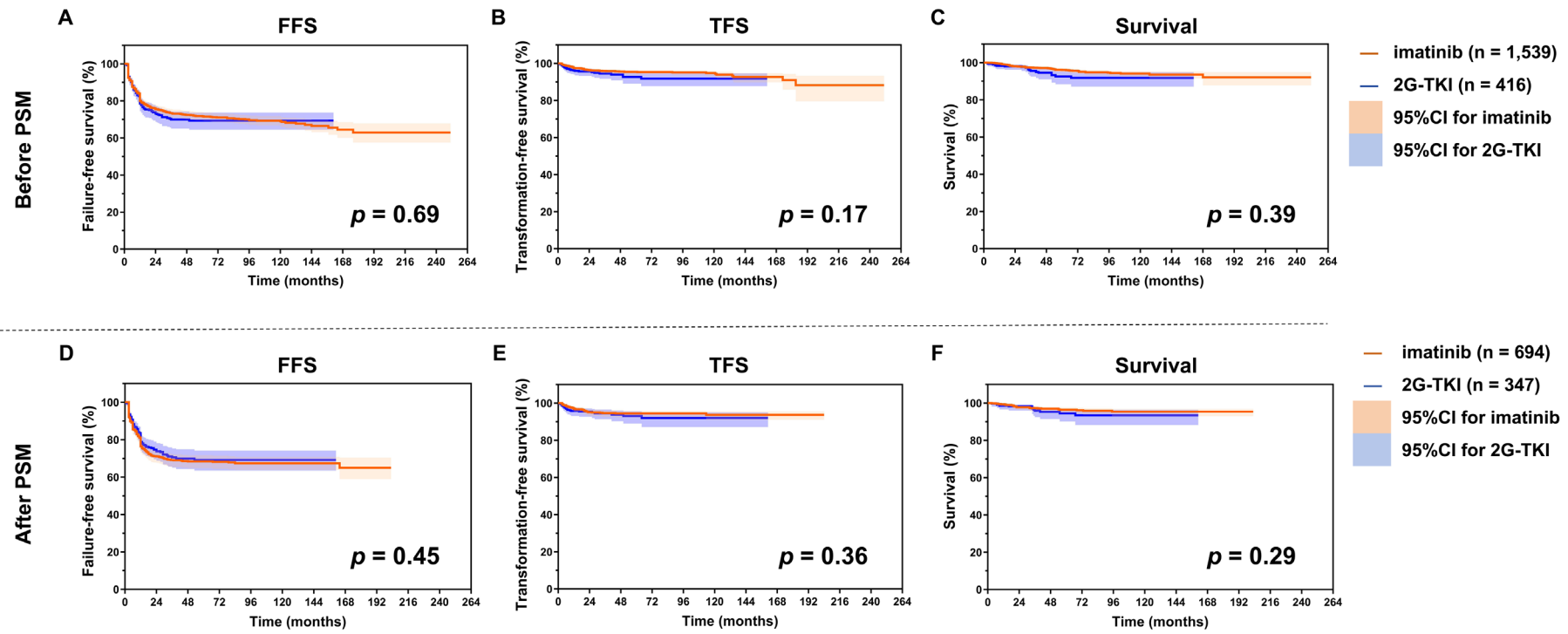
Supplement Figure 6. Cumulative incidences of molecular responses and probabilities of TFS, CML-related survival by the predictive model.

Supplement Figure 7. Cumulative incidences of therapy-failure by the Sokal and ELTS risk subgroups in the entire dataset.

Supplement Figure 8. The 1-, 3- and 5-year area under the receiver operating characteristic curve (AUROC) values of Sokal, ELTS and the therapy-failure models in the entire, imatinib and 2G-TKI cohorts.

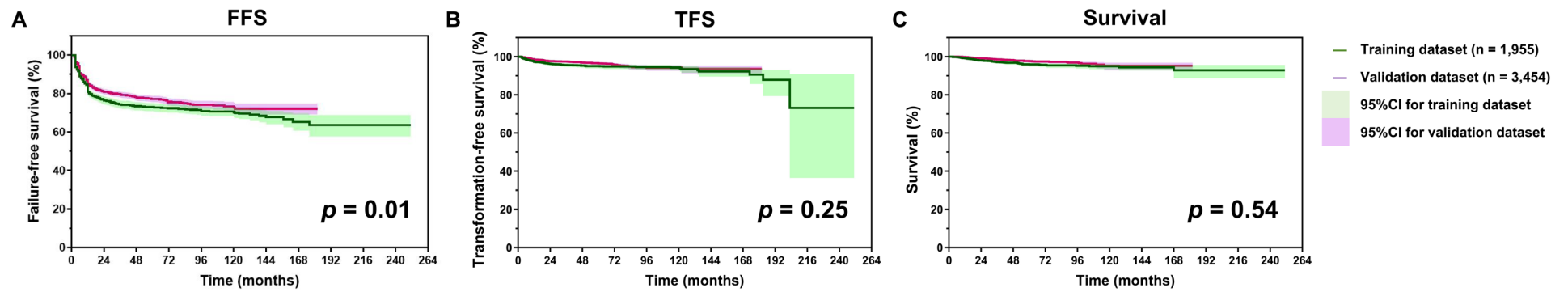
Supplement Figure 9. Decision curve analyses (DCA) of the predictive model, Sokal and ELTS scores for predicting the 1-, 3- and 5-year cumulative incidences of the therapy-failure in the entire, imatinib and 2G-TKI cohorts.

Supplement Figure 10. Comparison of the cumulative incidences of therapy-failure between subjects receiving initial 2G-TKI- and imatinib-therapy using the predictive model before and after propensity-score matching (PSM) analyses.



Supplement Figure 1. Comparison outcomes between imatinib and 2G-TKI cohorts in the training dataset before and after propensity-score matching (PSM) analyses.

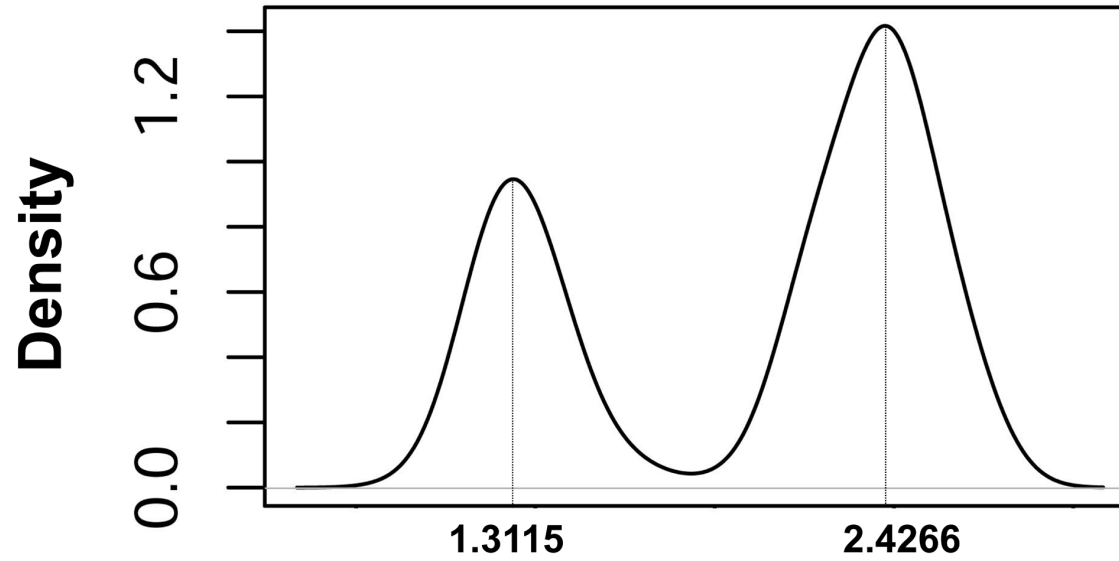
2G-TKI, the second-generation tyrosine kinase inhibitor; FFS, failure-free survival; TFS, transformation-free survival; Shaded area is the 95% Confidence Interval.



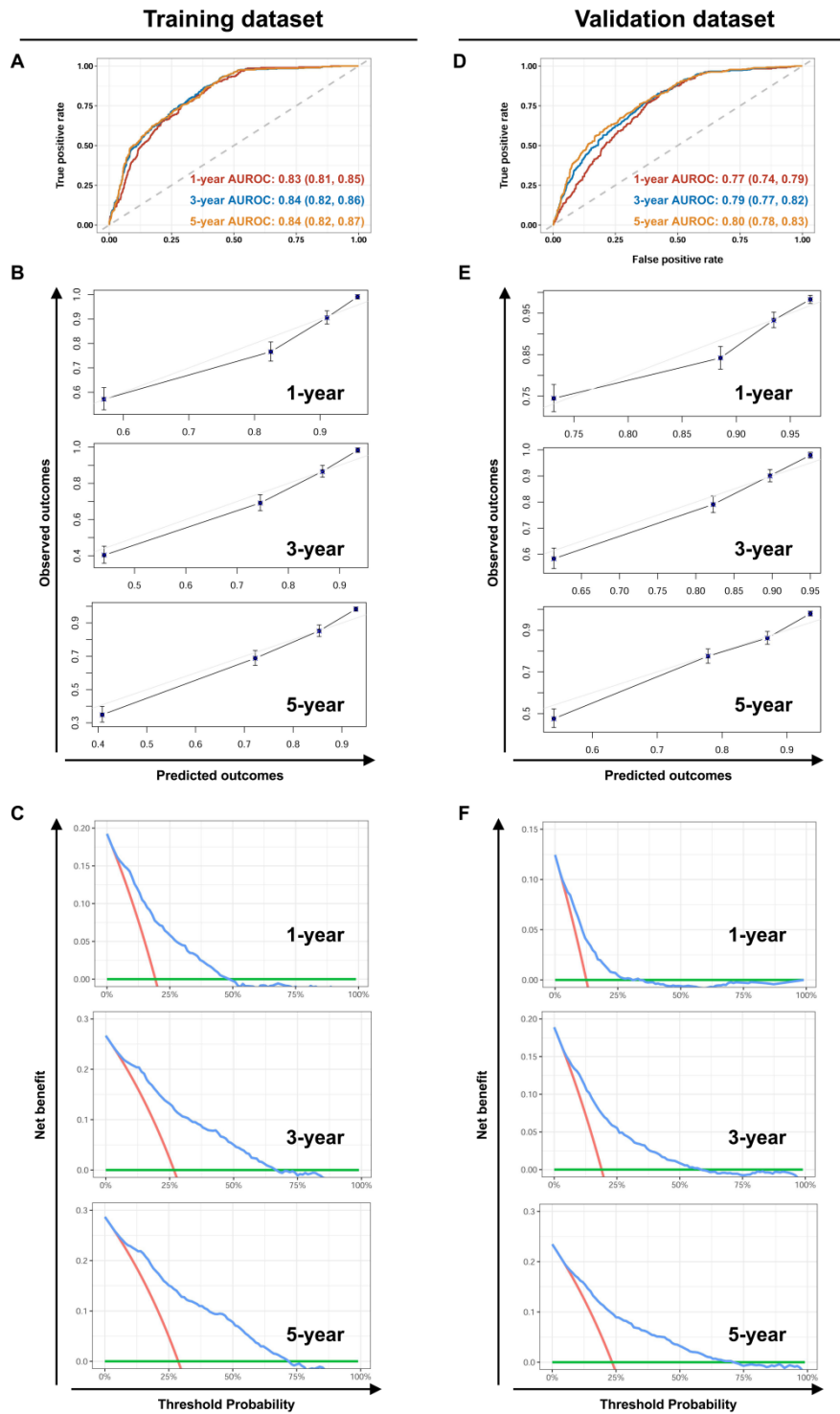
Supplement Figure 2. Comparison outcomes between training and validation datasets.

FFS, failure-free survival; TFS, transformation-free survival; Shaded area is the 95% Confidence Interval.

Kernel density estimation



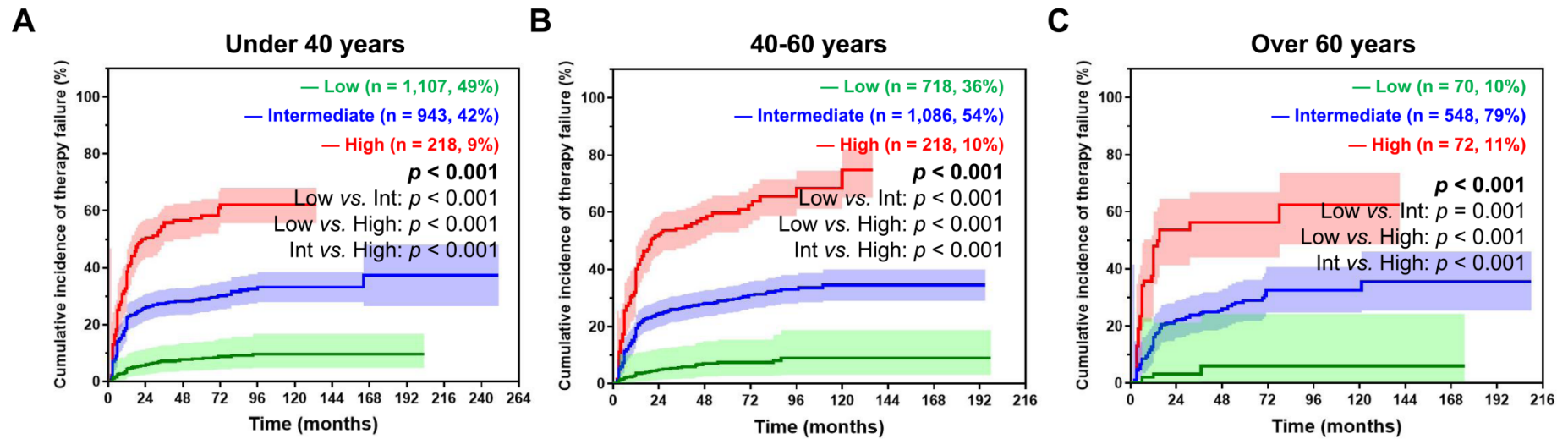
Supplement Figure 3. Kernel density plot of optimal cutoffs for the predictive model.



Supplement Figure 4. Performance of the predictive model for the cumulative incidence of therapy-failure.

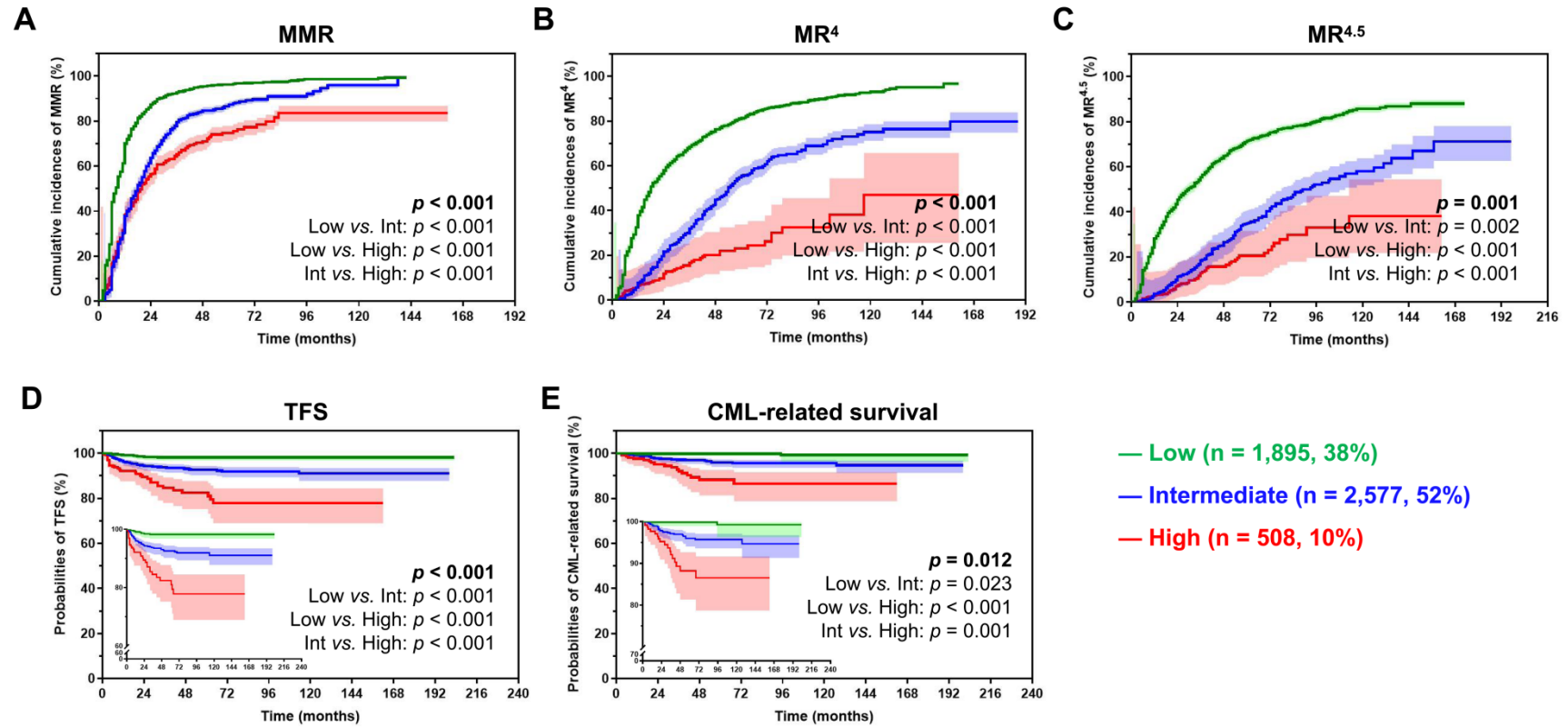
(A) AUROC curves of the predictive model for 1-, 3- and 5-year cumulative incidences of therapy-failure in the training dataset; (B) Calibration plots of the model for predicting 1-, 3- and 5-year cumulative incidences of

therapy-failure in the training dataset. Predicted probabilities of 1-, 3- and 5-year cumulative incidences of therapy-failure were plotted on the x-axis; actual cumulative incidences of therapy-failure were plotted on the y-axis. An ideal calibration plot is indicated by a 45° diagonal line; (C) Decision curve analysis of the predictive model for predicting the 1-, 3- and 5-year cumulative incidences of the therapy-failure in the training dataset; (D) AUROC curves of the predictive model for the 1-, 3- and 5-year cumulative incidences of therapy-failure in the validation dataset; (E) Calibration plots of the predictive model for predicting 1-, 3- and 5-year cumulative incidences of therapy-failure in the validation dataset. (F) Decision curve analysis of the predictive model for predicting the 1-, 3- and 5-year cumulative incidences of therapy-failure in the validation dataset. DCA plots of the “net benefit” against “threshold probabilities (P(t))”, assessing the clinical usefulness of the predictive model at appropriate thresholds for clinical use. Net benefit measures the trade-off between true- and false-positives in the prediction model at different threshold probabilities (P(t)). It is a sum of true- minus false-positive predictions weighted by the threshold probability as described in the following equation:
$$Net\ benefit = (true\ positive\ count / n) - (false\ positive\ count / n) \times (P(t)/1-P(t)).$$
 The threshold is a clinically derived value that varies depending on how risk averse the clinician is. It is a value where the clinician would be satisfied with the trade-off between the harm of delaying intervention (targeted follow-up) and unnecessary intervention. The x-axis is the threshold probability. In the risk model the cumulative incidence of the therapy failure is recorded as P(i). When P(i) reaches a certain threshold (scored as P(t)) an intervention is done. People will benefit from the intervention and there will be a detriment to people not taking the intervention. The Y-axis is the net benefit after the intervention minus the harm of not intervening.



Supplement Figure 5. Cumulative incidences of therapy-failure by different age subgroups.

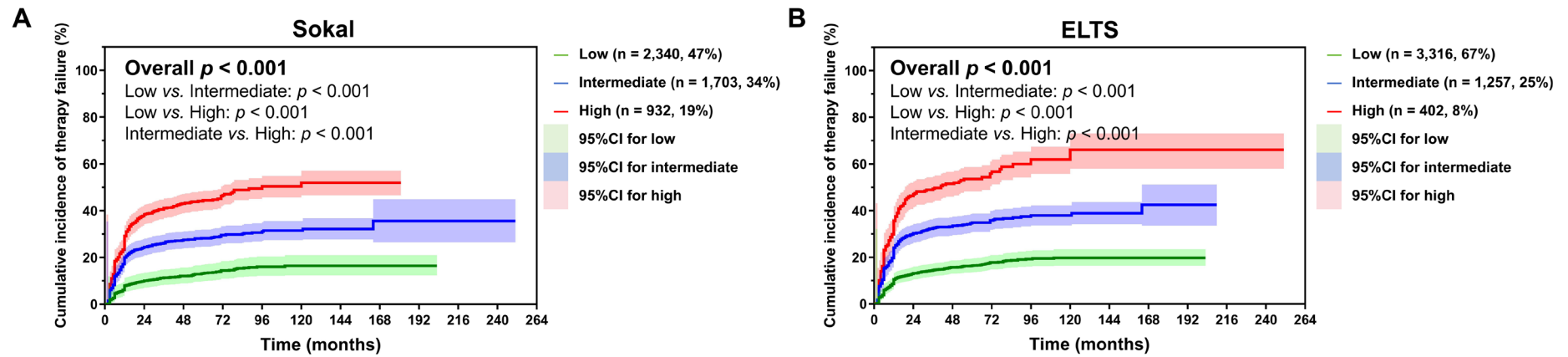
Shaded area is the 95% Confidence Interval.



Supplement Figure 6. Cumulative incidences of molecular responses and probabilities of TFS, CML-related survival by the predictive model.

MMR, major molecular response; MR⁴, molecular response 4; MR^{4.5}, molecular response 4.5; TFS, transformation-free survival.

Shaded area is the 95% Confidence Interval.



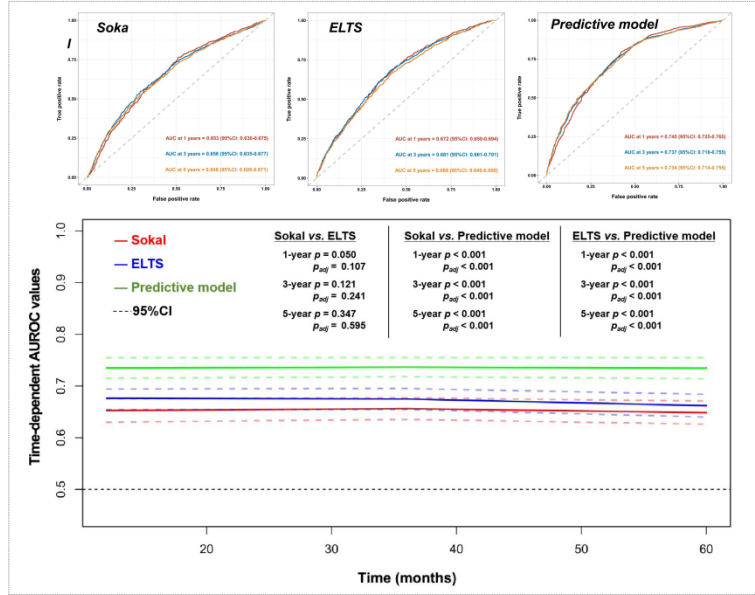
Supplement Figure 7. Cumulative incidences of therapy-failure by the Sokal and ELTS risk subgroups in the entire dataset.

ELTS, EUTOs long-term survival score; Shaded area is the 95% Confidence Interval.

(A) By Sokal risk score; (B) By ELTS score.

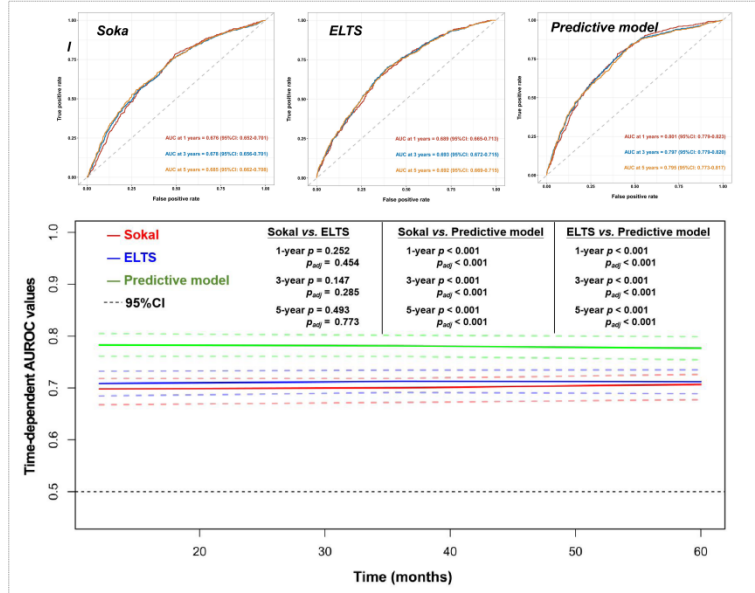
A

Entire cohort



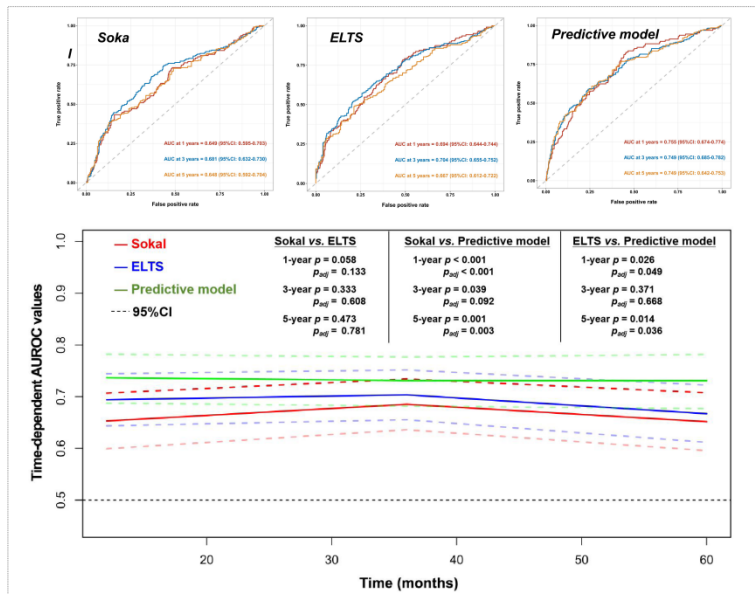
B

Imatinib cohort



C

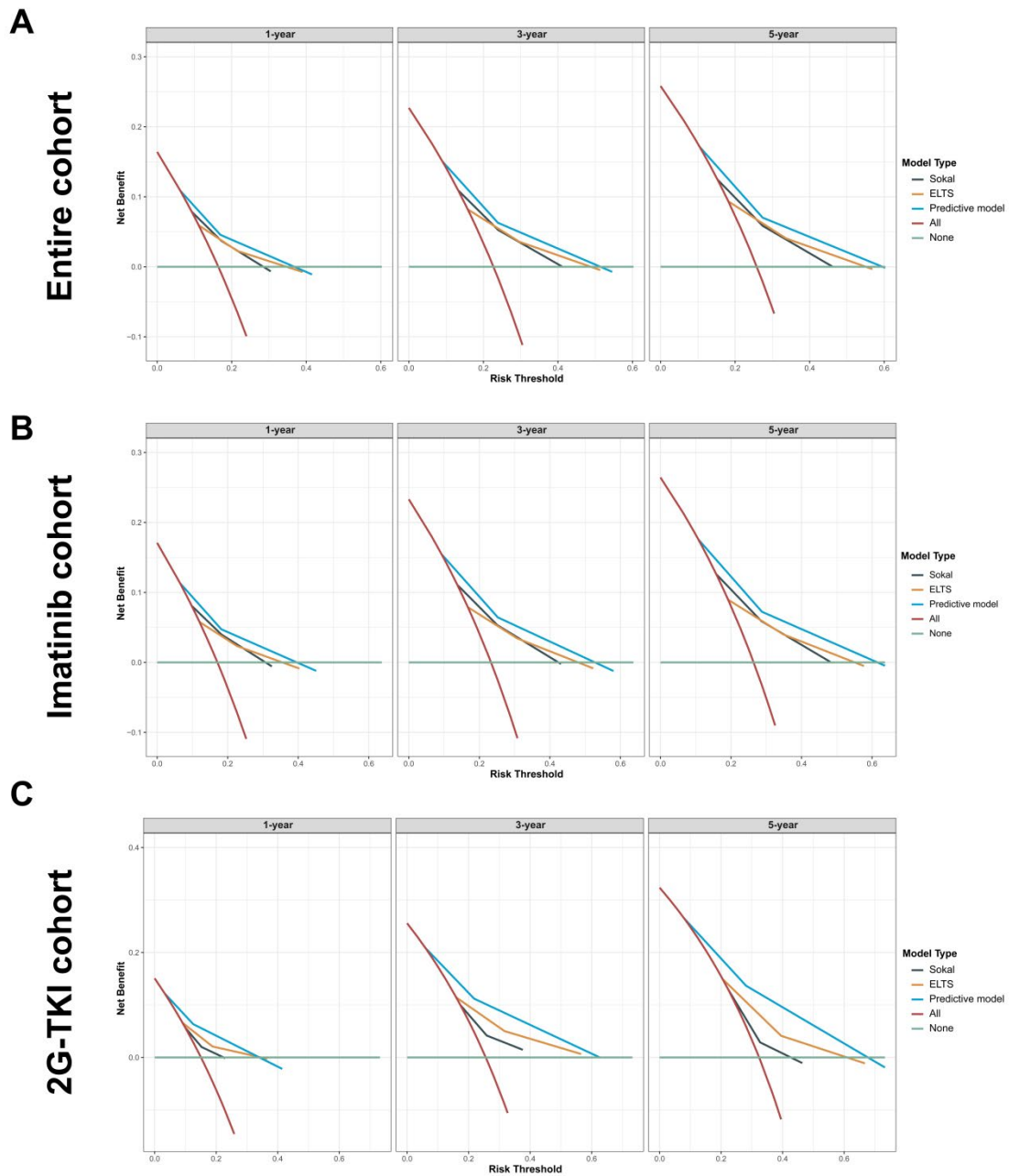
2G-TKI cohort



Supplement Figure 8. The 1-, 3- and 5-year area under the receiver operating characteristic curve (AUROC) values of Sokal, ELTS and the therapy-failure models in the entire, imatinib and 2G-TKI cohorts.

2G-TKI, the second-generation tyrosine kinase inhibitor.

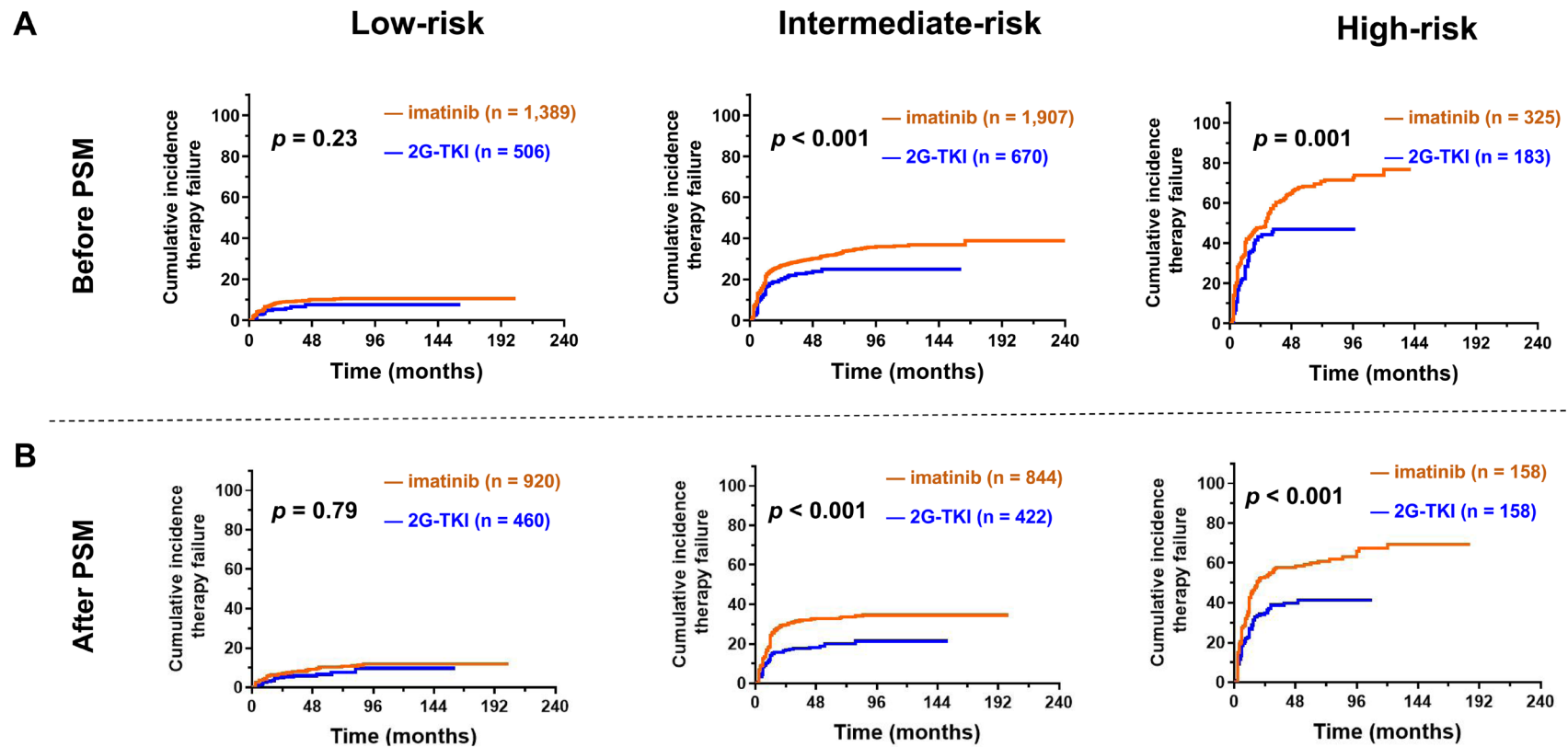
(A) Entire cohort; (B) Imatinib cohort; (C) 2G-TKI cohort.



Supplement Figure 9. Decision curve analyses (DCA) of the predictive model, Sokal and ELTS scores for predicting the 1-, 3- and 5-year cumulative incidences of the therapy-failure in the total, imatinib and 2G-TKI cohorts.

2G-TKI, the second-generation tyrosine kinase inhibitor.

(A) Entire cohort; (B) Imatinib cohort; (C) 2G-TKI cohort.



Supplement Figure 10. Comparison of the cumulative incidences of therapy-failure between subjects receiving initial 2G-TKI- and imatinib-therapy using the predictive model before and after propensity-score matching (PSM) analyses.

(A) Before PSM analyses; (B) After PSM analyses.