Supplementary Appendix

High throughput sequencing (HTS) methodology

High throughput sequencing (HTS) of a 37-gene panel (Table S1 below) was done on bone marrow (BM, n=370) or peripheral blood (PB, n=101) samples collected at inclusion in one central lab (Lille, University Hospital, Pr C. Preudhomme). Somatic mutations were identified by 2 orthogonal HTS methods. First, libraries were prepared using the Ampliseq technology and run on Ion Proton according to the manufacturer's instructions (ThermoFisher, Watham, MA, USA). Raw data were analyzed with two distinct softwares: Torrent Browser (Thermofisher) and SeqNext (JSI Medical System, Los Angeles, CA, USA). Second, librairies were prepared using the Haloplex Target Enrichment System (Agilent Technologies, Santa Clara, CA, USA) and run on MiSeq (Illumina, San Diego, CA, USA). Raw data were also analyzed on two distinct softwares: SureCall (Agilent Technologies) and SeqNext (JSI Medical System). A high depth of coverage (>1500x) was obtained for all genes analyzed with both HTS technologies, allowing detection of variants with a VAF (variant allele frequency) above 1%. Nonsense variants and frameshift insertions or deletions (indels) were always considered as somatic mutations. False sense variants and in frame indels were studied according to the human and public databases of somatic mutations or polymorphisms (SNP) and their VAF. Prediction of functional consequences of variants was performed using 6 prediction software programs: MAPP (Multivariate Analysis of Protein Polymorphism), PhD-SNP (Predictor of human Deleterious Single Nucleotide Polymorphism), PolyPhen-1, PolyPhen-2, SIFT (Sorting Intolerant From Tolerant), SNAP (Screening for Non-Acceptable Polymorphism).[1] Genotyping of *FLT3* internal tandem duplications (*FLT3*-ITD) was done by fragment analysis as previously published and expressed by the ratio ITD/wild type.[2] Because of technical limitations, the duplication of a guanine in position 1934 in *ASXL1* was investigated for all patients by fragment technique and confirmed by Sanger sequencing.[3] Finally, mutation in the *CEBPA* transcription factor gene was carried out using Sanger sequencing.[4] The subset of AML with sAML-like gene mutations was defined by mutation occurring in at least one of the genes listed in Table S2 below.[5]

Table S1. 37-gene panel

GENE	LOCUS	NM	COVERAGE
ASXL1	20q11.21	NM_015338	exons 11-12 ; exon 12 (AA 574-735) Fragment + Sanger
BCOR	Xp11.4	NM_001123385	exons 2-15
BCORL1	Xq26.1	NM_021946	exons 1-12
CALR	19p13.13	NM_004343.3	exon 9
CEBPA	19q13.11	NM_004364	exon 1 (Sanger)
CBL	11q23.3	NM_005188	exons 8-9
CSF3R	1p34.3	NM_172313.2	exons 14-18
DNMT3A	2p23.3	NM_022552	exons 2-23
ETV6	12p13.2	NM_001987	exons 1-8
EZH2	7q36.1	NM_004456	exons 2-20
FLT3-ITD	13q12.2	NM_004119	exons 14-15 (Fragment)
FLT3-TKD	13q12.2	NM_004119	exon 20
GATA2	3q21.3	NM_032638	exons 2-6
IDH1	2q34	NM_005896	exon 4
IDH2	15q26.1	NM_002168	exon 4
JAK2	9p24.1	NM_004972	exons 12 & 14
KIT	4q12	NM_000222	exons 8-11 & exon 17
KRAS	12p12.1	NM_033360	exons 2-3
MPL	1p34.2	NM_005373	exon 10
NIPBL	5p13.2	NM_133433	exons 2-47
NPM1	5q35.1	NM_002520	exon 11
NRAS	1p13.2	NM_002524	exons 2-3
PHF6	Xq26.2	NM_001015877	exons 2-10
PTPN11	12q24.13	NM_002834	exons 3 & 13
RAD21	8q24.11	NM_006265	exons 2-14
RIT1	1q22	NM_006912	exon 5
RUNX1	21q22.12	NM_001001890	exons 1-6
SETBP1	18q12.3	NM_015559	exon 4
SF3B1	2q33.1	NM_012433	exons 13-16
SMC1A	Xp11.22	NM_006306	exons 1-25
SMC3	10q25.2	NM_005445	exons 1-29
SRSF2	17q25.1	NM_003016	exon 1
STAG2	Xq25	NM_001042749	exons 3-35
TET2	4q24	NM_001127208	exons 3-11
TP53	17p13.1	NM_001126112	exons 3-11
U2AF1	21q22.3	NM_006758	exons 2 & 6
WT1	11p13	NM_024426	exons 7 & 9
ZRSR2	Xp22.2	NM_005089	exons 1-11

Table S2. sAML-like gene mutations in 471 patients tested

Gene	Locus	RefSeq	ALFA panel	Missing exons *	Mutation frequency, N (%)
ASXL1	20q11.21	NM_015338	exons 11-12	1-10 & 13	89 (19%)
SRSF2	17q25.1	NM_003016	exon 1	none	83 (18%)
STAG2	Xq25	NM_001042749	exons 2-35	none	39 (8%)
BCOR	Xp11.4	NM_001123385	exons 2-15	none	38 (8%)
U2AF1	21q22.3	NM_006758	exons 2 & 6	none	32 (7%)
EZH2	7q36.1	NM_004456	exons 2-20	none	26 (6%)
SF3B1	2q33.1	NM_012433	exons 13-16	1-12 & 17-25	23 (4%)
ZRSR2	Xp22.2	NM_005089	exons 1-11	none	10 (2%)

A total of 471 (93%) of the 509 patients were tested using a 37-gene myeloid panel. Presence of at least one gene mutation among this list of gene retained by Lindsley *et al.* was used to define sAML-like patients.[5]

References

1. Bendl J, Stourac J, Salanda O, et al. PredictSNP: robust and accurate consensus classifier for prediction of disease-related mutations. *PLoS Comput Biol*. 2014;10(1):e1003440.
2. Renneville A, Boissel N, Nibourel O, et al. Prognostic significance of DNA methyltransferase 3A mutations in cytogenetically normal acute myeloid leukemia: a study by the Acute Leukemia French Association. *Leukemia*. 2012;26(6):1247-1254.
3. Duployez N, Marceau-Renaut A, Boissel N, et al. Comprehensive mutational profiling of core binding factor acute myeloid leukemia. *Blood*. 2016;127(20):2451-2459.
4. Preudhomme C, Sagot C, Boissel N, et al. Favorable prognostic significance of *CEBPA* mutations in patients with de novo acute myeloid leukemia: a study from the Acute Leukemia French Association (ALFA). *Blood*. 2002;100(8):2717-2723.
5. Lindsley RC, Mar BG, Mazzola E et al. Acute myeloid leukemia ontogeny is defined by distinct somatic mutations. *Blood*. 2015;125(9):1367-1376.

European LeukemiaNet (ELN) group distributions

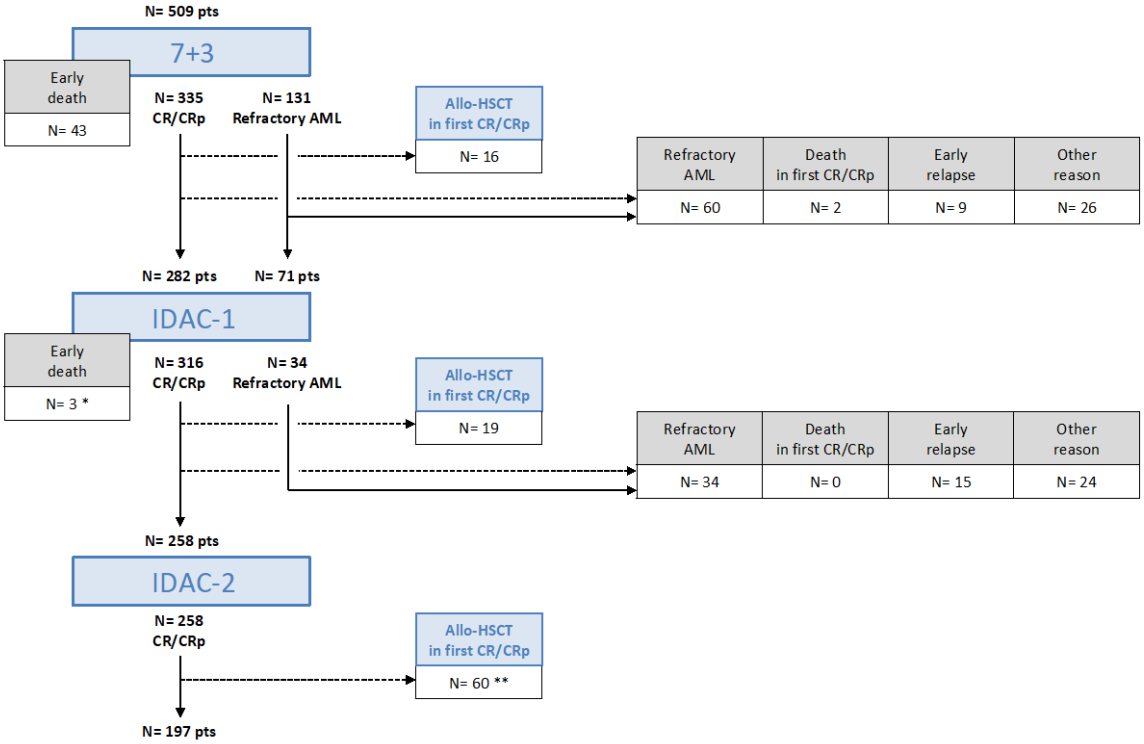
Cytogenetic and genomic characteristics were centrally reviewed during the study in order to classify the patients within the ELN-2010 classification subgroups,[1] used for allogeneic HSCT eligibility in first remission. Standard Sanger PCR was used to detect *CEBPA* and *NPM1* gene mutations and fragment analysis to determine *FLT3*-ITD allelic ratios. Biallelic *CEBPA* gene mutations only were retained to classified cases as of favorable risk. Overall, 74 (15%), 312 (61%) and 86 (17%) patients were prospectively classified as with favorable-, intermediate- and adverse-risk AML, respectively. In order to classify all patients for transplantation *versus* no-transplantation indication, the remaining 37 patients (7%) were prospectively classified as follows: 29 patients with cytogenetic failure and no favorable gene mutation were allocated to the intermediate-risk subgroup, while 8 patients with less than 20 normal metaphases, no abnormal metaphase and favorable gene mutations were allocated to the favorable-risk group.

At the end of the study, gene mutation patterns, including *ASXL1*, *RUNX1* and *TP53* mutational status, were retrospectively determined by next-generation sequencing in 471 (93%) of the 509 patients using a 37-gene myeloid panel, allowing reclassification of 493 of the 509 (97%) study patients within the recently updated ELN 2017 classification.[2]

References

1. 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.
2. Döhner H, Estey E, Grimwade D et al. Diagnosis and management of AML in adults: 2017 ELN recommendations from an international expert panel. *Blood*. 2017;129(4):424-447.

CONSORT diagram

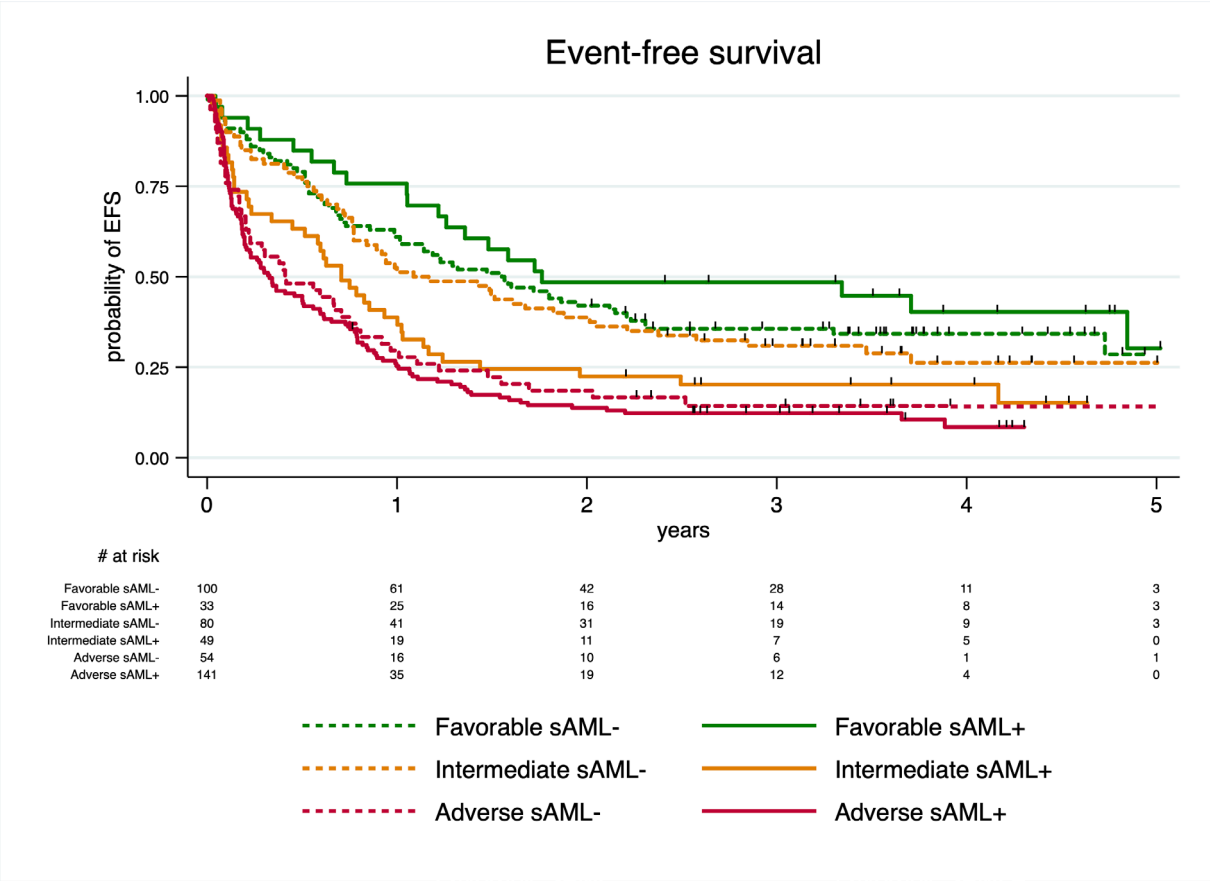


*: these 3 patients received the first IDAC course as salvage, as not in CR/CRp after the first induction course; **: including 2 patients with favorable-risk AML, thus not eligible for HSCT in first remission per protocol.

In the original version of the protocol, four additional 1+5 low-intensity maintenance courses were planned after the second IDAC course in non-transplanted patients. Fifty-six patients only actually received these low-intensity courses, as such maintenance was abandoned by amendment on February 2013 due to a poor adherence of physicians in most centers. By the same amendment, cytarabine dosage was reduced to 1g/m^2/12h during consolidation courses above 70 years of age, instead of 75 years of age as originally recommended. Administration of maintenance courses, however, did not influence neither relapse incidence nor survival rates (data not shown).

Event-free survival according the presence of sAML-like gene mutations in the three ELN-2017 risk subgroups, separately

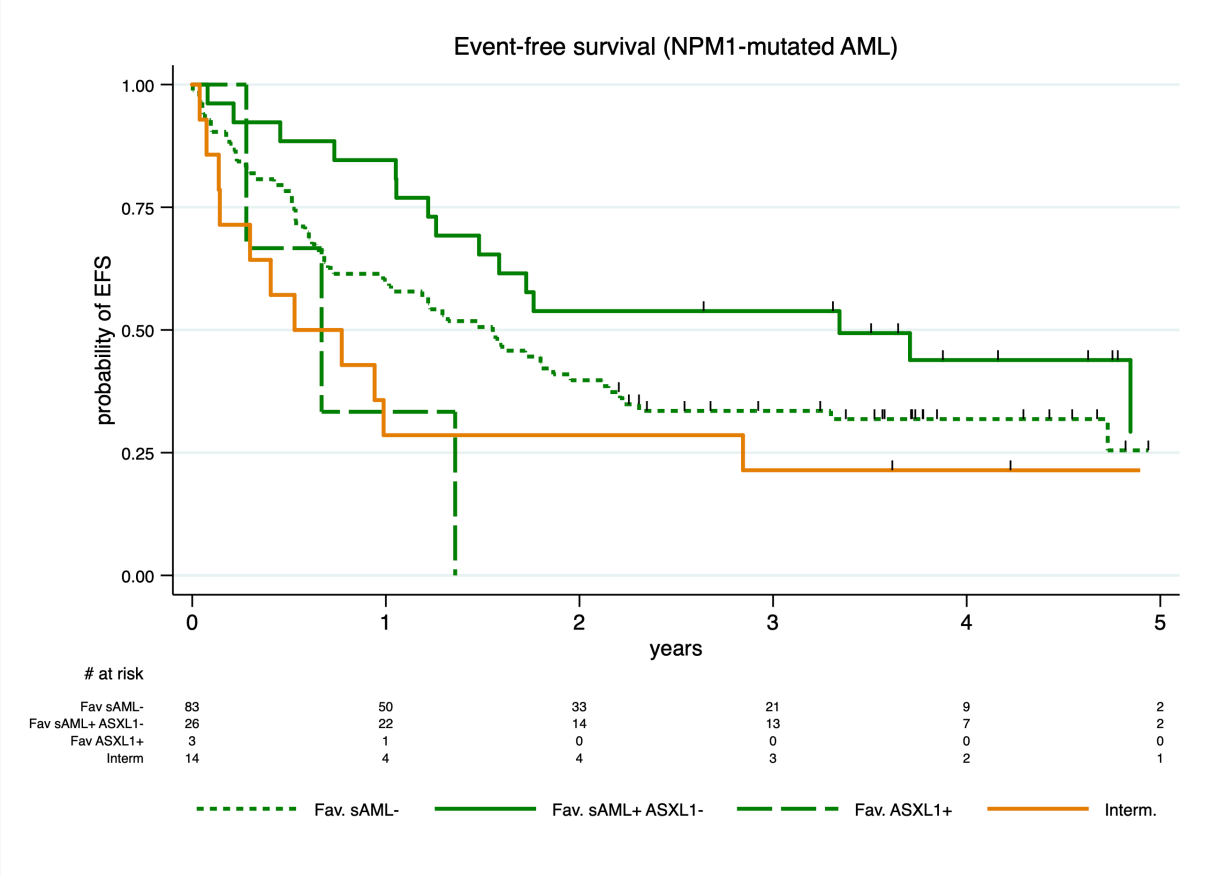
Figure S1



EFS according to sAML-like mutations in the three ELN-2017 risk subgroups, separately. The presence of sAML-like gene mutations did not influence EFS in the favorable-risk (HR, 0.78 [95% CI, 0.47-1.28]; P= 0.32) nor in the adverse-risk (HR, 1.12 [95% CI, 0.80-1.57]; P= 0.51) subgroup, while in the intermediate-risk subgroup these mutations (by definition other than *ASXL1* mutation here) retained their prognostic value (HR, 1.52 [95% CI, 1.01-2.28]; P= 0.044).

Event-free survival according the presence of sAML-like gene mutations in *NPM1*-mutated AML

Figure S2



EFS according to sAML-like mutations in the *NPM1*-mutated AML subgroup. Among the 127 *NPM1*-mutated AML cases tested for sAML-like mutations, 112, 14 and 1 could be classified in the ELN-2017 favorable, intermediate and adverse risk subgroup, respectively. Among these 112 favorable-risk cases, one or more sAML-like gene mutations were detected in 29 cases (including 3 *ASXL1* mutations). Among the 14 intermediate-risk cases (all with high ratio *FLT3*-ITD mutation), sAML-like gene mutations were only detected in 2 patients. As illustrated in the Figure S2 above, among the 112 favorable-risk patients, the presence of sAML-like gene mutations other than ASXL1 did not alter EFS (HR, 0.64 [95% CI, 0.36-1.13]; p= 0.12).

Allogeneic HSCT in first remission

Patients eligible for HSCT

According to the study design, 305 CR/CRp patients with ELN 2010 intermediate or adverse risk AML were eligible for allogeneic HSCT. A total of 93 eligible patients were actually transplanted in first remission, including only 3 patients older than 70 years.

The role of HSCT was thus evaluated in the 211 eligible patients aged 70 years or less, 90 of them being transplanted (43%). Characteristics of the 90 *versus* the 121 non-transplanted patients are shown in the Table S3 below. Notwithstanding a lower median age of the transplanted patients, other characteristics of the transplant and no-transplant cohorts were well-balanced. In the 90 transplanted patients, median time from remission to transplant was 113 days (IQR, 80-134). Hematopoietic stem cell source was a sibling donor, a matched unrelated donor, a related haplo-identical donor or cord blood units in 28, 50, 8 and 4 patients, respectively. Conditioning regimen, not specified by the protocol was fludarabine/busulfan (FB)-based in 66 patients, FB-TBI in 12 patients, sequential FLAMSA-type conditioning in 12 patients, and a more intensive FB-based conditioning in 1 patient.

With a median follow-up of 3.8 years, 124 out of these 211 patients aged 70 years old or less and eligible for HSCT in first remission eventually relapsed, including 26 relapses in transplanted patients. Forty-three patients received an intensive salvage treatment, 37 (30%) achieved a second remission and 18 (15%) were transplanted after relapse, including 13 transplantation in second remission. Overall, median EFS was 17.2 months (95% CI, 12.8-20.7) and 4-year EFS was estimated at 29% (95% CI, 23-36). Median OS was 33.2 months (95% CI, 24.8-46.1) and 4-year OS was estimated at 42% (95% CI, 25-50). Median RFS was 15.9 months (95% CI, 11.3-19.1) and 4-year RFS was estimated at 29% (95% CI, 23-36). In patients who received HSCT, cumulative incidences or relapse (CIR), respectively non-relapse mortality (CINRM) after HSCT, was estimated from the date of transplant, non-relapse related death, respectively relapse, being considered as a competing event. At 2 years, cumulative incidences of relapse and non-relapse mortality were 23% (95% CI, 16-34) and 19% (95% CI, 12-29), respectively.

Table S3. Characteristics of patients aged 70 years old or less and eligible for HSCT

	No HSCT in first remission	**HSCT in first remission**	**P value**
Patients, N	121	90	-
Gender, M/F	67/54	53/38	0.78
Median age (range)	66 years (60-70)	64 years (60-70)	0.0037
ECOG-PS, 0/1/2+/NA	50/57/11/3	47/35/8/0	0.32
HCT-CI, 0/1/2/3+/NA	53/28/13/25/2	52/14/8/13/3	0.11
Secondary AML, N (%)	12 (10%)	17 (19%)	0.071
Therapy-related AML, N (%)	7 (6%)	3 (3%)	0.52
ELN-2010 risk group, N (%)	-	-	0.51
Favorable *	2 (2%)	0 (0%)	-
Intermediate	95 (78%)	75 (83%)	-
Adverse	24 (20%)	15 (17%)	-
ELN 2017 risk group, N (%)	-	-	0.16
Favorable risk *	26 (21%)	12 (13%)	-
Intermediate risk	46 (38%)	37 (41%)	-
Adverse risk	43 (36%)	40 (45%)	-
Not available	6 (5%)	1 (1%)	-
SR/HR risk groups, N classifiable **	-	-	0.56
Standard-risk (SR)	52	37	-
High-risk (HR)	54	47	-

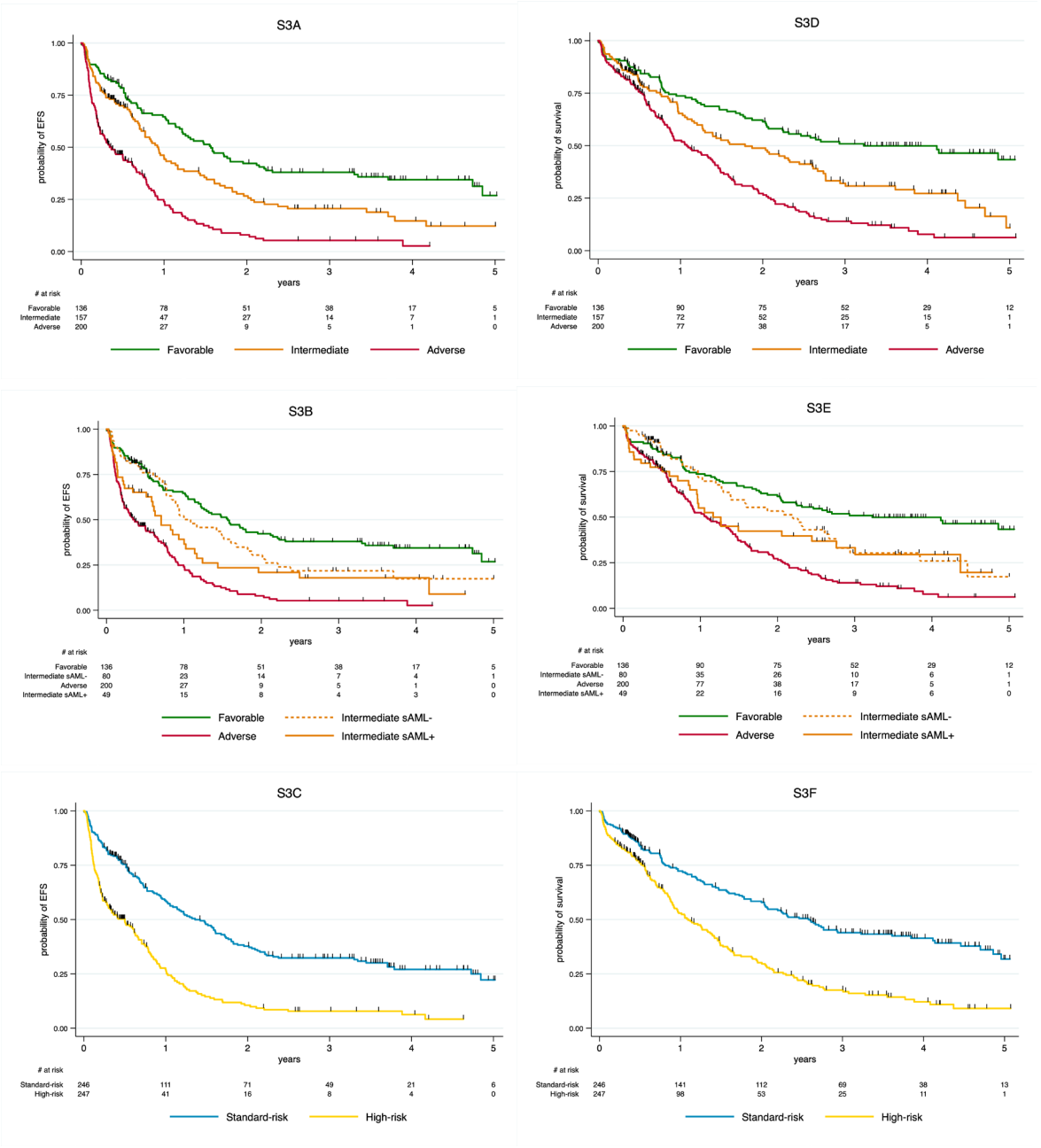
*: the 2 patients with ELN 2010 favorable-risk AML were eligible for transplantation, as they needed two courses to reach remission; of note, the 36 additional patients with ELN 2017 favorable-risk AML were not representative of a general cohort of favorable-risk patients, as they could not have ELN 2010 favorable-risk AML to be eligible for transplantation according to the protocol. **: 190 of these 211 patients were eventually classified in the standard-risk (ELN 2017 intermediate risk without sAML-like gene mutation or ELN 2017 favorable risk AML) or high-risk (ELN 2017 intermediate risk with sAML-like mutation or ELN 2017 adverse risk AML) subgroup.

Transplant procedures

In the 93 eligible patients who received allogeneic HSCT in first remission, the median time from remission to transplant was 113 days (IQR, 80-134). Hematopoietic stem cell source was a sibling donor, a matched unrelated donor, a related haplo-identical donor or cord blood units in 28, 52, 8 and 5 patients, respectively. Conditioning regimen, not specified by the protocol was fludarabine/busulfan (FB)-based in 68 patients, FB-TBI in 13 patients, sequential FLAMSA-type conditioning in 12 patients, and a more intensive FB-based conditioning in 1 patient. In patients who received HSCT, 2-year cumulative incidences of relapse and non-relapse mortality were 25% (95% CI, 17-35) and 19% (95% CI, 13-29), respectively.

Prognostic impact of sAML-like gene mutations, censoring the patients allografted in first remission at transplant time

Figure S3



S3A. EFS according to the three ELN-2017 risk subgroups (P<0.001). At 2 years, EFS was estimated at 42.3% (95% CI, 33.5-50.8), 26.7% (95%CI, 18.7-35.2) and 8.0% (95% CI, 4.0-13.7) in the favorable-, intermediate- and adverse-risk, respectively. At 4 years, EFS was estimated at 34.5% (95% CI, 25.9-43.1), 14.7% (95%CI, 7.9-23.5) and 2.7% (95% CI, 0.4-9.6) in the favorable-, intermediate- and adverse-risk, respectively.

S3B. EFS according to sAML-like mutations in the ELN-2017 intermediate-risk subgroup. The presence of sAML-like gene mutations did not significantly influence EFS in the ELN-2017 intermediate-risk subgroup, even if a trend for a worse EFS was observed (HR, 1.44 [95% CI, 0.93-2.24]; P= 0.10).

S3C. EFS according to the newly defined HR/SR risk groups. EFS was significantly reduced in the HR group (HR, 2.41 [95% CI, 1.93-3.01]; p<0.001). At 2 years, EFS was estimated at 37.7% (95% CI, 31.0-44.4) in the SR group, as compared to 10.6% (95%CI, 6.4-15.9) in the HR group. At 4 years, EFS was estimated at 27.1% (95% CI, 20.6-34.0) in the SR group, as compared to 6.2% (95%CI, 2.8-11.6) in the HR group.

S3D. OS according to the three ELN-2017 risk subgroups (P<0.001). At 2 years, OS was estimated at 61.4% (95% CI, 52.3-69.3), 48.8% (95%CI, 39.5-57.5) and 27.2% (95% CI, 20.3-34.5) in the favorable-, intermediate- and adverse-risk, respectively. At 4 years, OS was estimated at 49.9% (95% CI, 40.6-58.4), 27.2% (95%CI, 18.5-36.7) and 7.8% (95% CI, 3.6-14.2) in the favorable-, intermediate- and adverse-risk, respectively.

S3E. OS according to sAML-like mutations in the ELN-2017 intermediate-risk subgroup. The presence of sAML-like gene mutations did not significantly influence OS in the ELN-2017 intermediate-risk subgroup (HR, 1.26 [95% CI, 0.79-2.06]; P= 0.32).

S3F. OS according to the newly defined HR/SR risk groups. OS was significantly reduced in the HR group (HR, 2.14 [95% CI, 1.69-2.71]; p<0.001). At 2 years, OS was estimated at 57.9% (95% CI, 50.8-64.3) in the SR group, as compared to 30.2% (95%CI, 23.7-36.8) in the HR group. At 4 years, OS was estimated at 41.4% (95% CI, 34.2-48.5) in the SR group, as compared to 12.1% (95%CI, 7.4-18.1) in the HR group.

Graphical representation of the HSCT effect

Figure S4A. Simon-Makuch plots for OS from remission by HSCT in SR patients

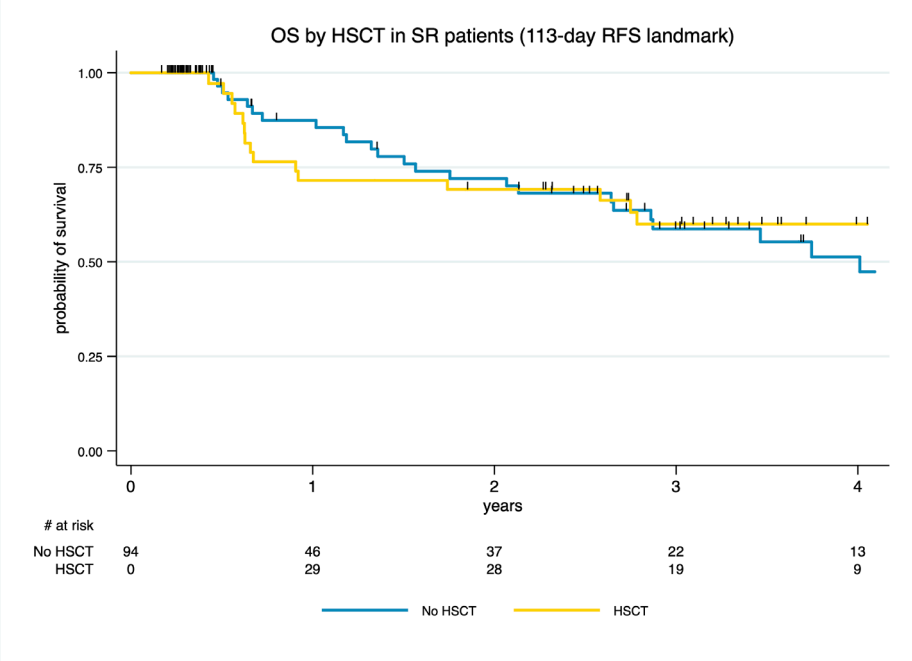
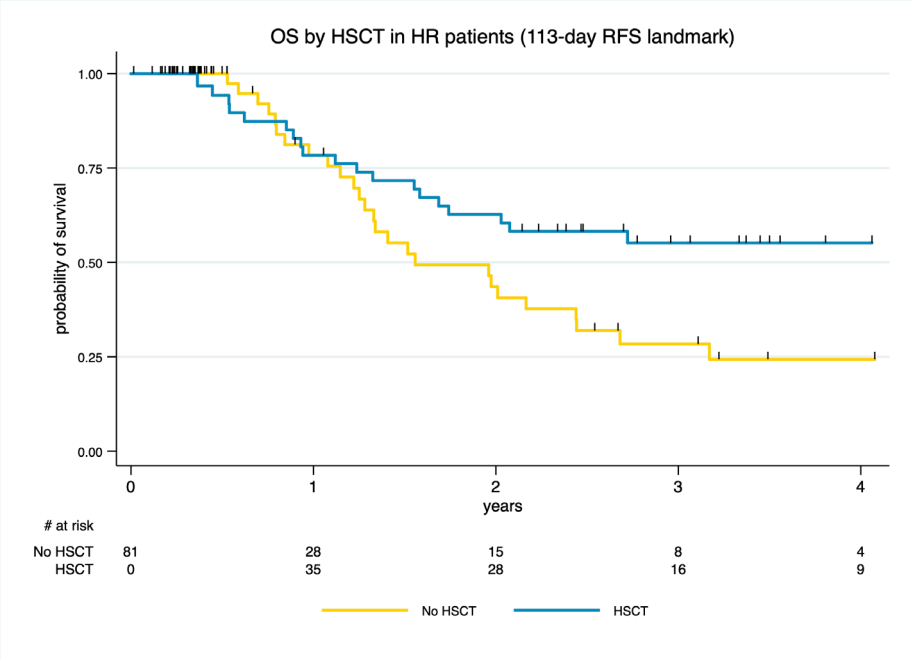


Figure S4B. Simon-Makuch plots for OS from remission by HSCT in HR patients



Multivariable analysis

Table S4 below summarizes the results of multivariable prognostic analysis for OS from remission, included HSCT in first remission as a time-dependent variable. As shown, transplantation was still significantly associated with prolonged OS from remission in patients with high-risk AML, while not in those with standard-risk AML.

Table S4. Multivariable analysis of HSCT effect on OS from remission

	HR	95% CI	P values
Patients aged 60 to 70 years old eligible for HSCT			
All patients	-	-	-
HSCT in first remission	0.61	0.38-0.98	0.041
High-risk *versus* standard-risk AML	1.78	1.13-2.80	0.013
Age	1.03	0.95-1.11	0.50
ECOG-PS ≥2	2.43	1.11-5.28	0.026
HCT-CI ≥3	1.34	0.77-2.32	0.30
WBC ≥50 G/L	1.20	0.60-2.42	0.61
Clinically defined sAML	0.88	0.42-1.84	0.74
Patients with standard-risk AML	-	-	-
HSCT in first remission	0.89	0.45-1.78	0.75
Age	1.04	0.93-1.17	0.44
ECOG-PS ≥2	0.53	0.12-2.39	0.41
HCT-CI ≥3	0.80	0.35-1.83	0.60
WBC ≥50 G/L	0.86	0.37-2.02	0.73
Clinically defined sAML	0.73	0.16-3.27	0.68
Patients with high-risk AML	-	-	-
HSCT in first remission	0.43	0.22-0.85	0.015
Age	0.99	0.89-1.10	0.87
ECOG-PS ≥2	10.1	4.03-30.0	<0.001
HCT-CI ≥3	1.67	0.77-3.59	0.19
WBC ≥50 G/L	6.31	1.69-23.5	0.006
Clinically defined sAML	0.92	0.37-2.25	0.85

Extended Cox models including allogeneic HSCT in first remission as a time-dependent covariate were performed in patients aged 60 to 70 years old eligible for HSCT as defined by the protocol, using a 113-day RFS landmark. HR, hazard ratio; CI, confidence interval; ECOG-PS, ECOG performance status; HCT-CI, hematopoietic cell transplantation comorbidity index; WBC, white blood cell count.

List of investigators in the Acute Leukemia French Association (ALFA)

C. Gardin, T. Braun, B. Papoular, S. Brechignac, J. Soret, G. Edouart, A. Douay, V. Eclache, R. Letestu (Hôpital Avicenne, Bobigny) ; JP. Marolleau, B. Gruson, D. Lebon, A. Charbonnier, V. Harrivel, A. Aleme, A. Hubert (CHU, Amiens); A. Al Jijakli, D. Chaoui, P. Genet, R. Skhiri, T. Guerekobaya, S. Talfi, M. Dubief (CH, Argenteuil); B. Choufi, M. Barry, M. Brument, J. Monvoisin, A. Ducrocq (CH, Boulogne/Mer); O. Reman, S. Chantepie, L. Damaj Ghandi, M. Macro, S. Cheze, H. Johnson Ansah, JB. Mear, D. Naguib, V. Salaun, N. Cornet, E. Marin (CHU, Caen); JV. Malfuson, J. Konopacki, A. Segot, F. de Charry, V. Foissaud, D. Bories (Hôpital Percy, Clamart); C. Salanoubat, S. Haiat, A. Devidas, B. Joly, C. Petitdidier, S. Cereja, H. Mossafa, I. Lemaire, P. Quillet, N. Hadjadj, (CH, Corbeil); C. Pautas, C. Cordonnier, S. Maury, A. Toma, Y. Hicheri, D. Bories, O. Wagner-Ballon, L. Cabanne, Y. Naitsidenas (Hôpital Henri Mondor, Créteil); M. Bemba, F. Bonnevie, M. Wetterwald, C. Delattre, C. Roche-Lestienne, V. Paquez (CH, Dunkerque); S. de Botton, JH. Bourhis, JB. Micol, N. Auger, V. Saada, L. Amghar (Institut Gustave Roussy, Villejuif); B. Quesnel, C. Berthon, H. Debarri, MO. Petillon, C. Roche-Lestienne, C. Preudhomme, O. Nibourel, C. Roumier, F. Dumezy, C. Rodriguez, C. Frimat, H. Djeda (CHU, Lille); P. Turlure, S. Girault, MP Gourin, P Chaury, MJ. Rapp, S. Moreau, L. Remenieras N. Gachard, F. Trimoreau, J. Abraham, M. Touati, J. Feuillard, E. Guerin, C. Philippon (CHU, Limoges); X. Thomas, F. Nicolini, L. Gilis, S. Ducastelle-Lepretre, L. Lebras, E. Wattel, F. Baracco, E. Paubelle, M. Balsat, MV. Larcher, H. Labussière, M. Michallet, S. Hayette, A. Plesa, D. Manzoni, I. Tigaud, M. Elhamri, A. Deloire, O. Le Borgne (Hôpital Lyon Sud, Lyon); J. Frayfer, W. Abarah, L. Fouillard, A. Cung, V. Gobert, MA. Ahui Brou (CH, Meaux); L. Gastaud, A. Thyss, F. Peyrade, S. Raynaud, I. Arnoux, C. Debaigt, A. Gama (Centre Lacassagne, Nice); T. Cluzeau, L. Manonne, M. Loschi, S. Raynaud, C. Hathroubi (CHU, Nice); O. Hermine, A. Marçais, F. Suarez, R. Delarue, C. Brouzes, N. Chapuis, T. Saheb (Hôpital Necker, Paris); H. Dombret, E. Raffoux, N. Boissel, E. Lengliné, F. Rabian, AM. Ronchetti, P. Fenaux L. ades, M. Sebert, C. Bally, R. Rahme, O. Maarek, S. Mathis, A. Raimbault, K. Celli-Lebras, R. Grapin, F. Hilaire, AS. Debrie, L. Amghar, I. Chouikh (Hôpital Saint-Louis, Paris); R. Benramdane, I. Vaida, H. Gonzalez, D. Mallet, C. Terré, V. Barriere, M. Amirault, J. Limea (CH, Pontoise); I. Plantier, M. Wemeau, L. Detourmignies, Dervite, A. Fawaz, C. Ghevaert, K. Dernivoix, C. Trouillet, Q. Dhalluin (CH, Roubaix); H. Tilly, E. Lemasle, N. Contentin, A. Stamatoulas, F. Jardin, S. Lepretre, P. Lenain, C.

Boutet, E. Bera, S. Vaudaux, V. Bayon, Y. Atia (Centre Henri Becquerel, Rouen); J. Vargaftig, S. Glaisner, C. Soussain, C. Terré, H. Bennani, V . Bardet, P. Vavasseur, (Centre Rene Huguenin, Saint-Cloud); P. Rousselot, H. Farhat, S. Ghez, J. Lambert, S. Rigaudeau, AL. Taksin, C. Terré, I. Garcia, S. Raggueneau, C. Fandomtchomgouo, C. Constant (CH, Versailles); J. Fernandes, N. Cambier, JP. Pollet, M. Simon, S. Tricot, A. Daudignon, H. Bisiau, C. Hemar, M. Fery (CH, Valenciennes), C. Rose, B. Carpentier, L. Pascal, N. Cambier, A. Charpentier, S. Waucampt, D. Brzyski, (CH, Lille Saint Vincent); GM. Pica, F. Prieur, A. Courouau (CH Métropole Savoie, Chambéry).