Supplemental Tables and Appendix

Supplemental Table 1. Chromosomal Translocations with Higher Prevalence in Pediatric AML than in Adult AML (Data Tabulated by Betsy Hirsch, Susana Raimondi, Soheil Meschinchi, Nyla Heerema and Andrew J. Carroll).

Translocation	**Gene Fusions**	**Frequency in Children /Frequency in Adults**	Age Group Predilection	**Comments/ Prognosis**	References
t(1;22)(p13.3;q13.1)	*RBM15(OTT)-MKL1(MAL)*	0.8%/0%	Infants	AMKL – FAB M7/ Intermediate	[1],[2]
t(7;12)(q36.3;p13.2)	*MNX1-ETV6*	0.8%/<0.5%	Infants	+19 seen as secondary abnormality Adverse	[3], [4], [5]
t(8;16)(p11.2;p13.3)	*KAT6A-CREBBP*	0.5%/<0.5%	Infants and children	Can spontaneously remit in infancy; intermediate prognosis in later childhood	[6]
t(6;9)(p23;q34.1)	*DEK-NUP214*	1.7%/1%	Older children; rare in infants	Adverse; 65% with *FLT3*-ITD	[7] [8]
11q23.3	*KMT2A* (*MLL*) translocated	25%/5-10%	Infant 50%	Prognosis dependent on the partner gene	[9],[10]
t(9;11)(p21.3;q23.3)	*KMT2A-MLLT3*	9.5%/2%	Children	Intermediate	[11]

t(10;11)(p12;q23.3)	*KMT2A-MLLT10*	3.5%/1%	Children	Include subtle and cryptic *KMT2A* rearrangements/ Adverse	[11]
t(6;11)(q27;q23.3)	*KMT2A-MLLT4*	2%/<0.5%	Children	Adverse	[11]
t(1;11)(q21;q23.3)	*KMT2A-MLLT11*	1%/<0.5%	Children	Favorable	[11]
Cryptic Chromosomal Translocations					
t(5;11)(q35.3;p15.5)	*NUP98-NSD1*	7%/3% 16% of *FLT3*-ITD patients	Older children and young adults	Adverse; 80% with *FLT3*-ITD. In combination associated with induction failure	[12],[13],[14]
inv(16)(p13.3q24.3)	*CBFA2T3-GLIS2*	3%/0%	10% of Infants, 20% of FAB M7	Adverse	[15],[16]
t(11;12)(p15.5;p13.5)	*NUP98-KDM5A*	3%/0%	Children <5 years 10% of FAB M7	Intermediate	[14],[17]

Supplemental Table 2. Molecular genetic alterations affecting clinical outcome of AML patients in specific cytogenetic groups (prepared by Krzysztof Mrózek).

Molecular Genetic Alteration	**Cytogenetic group**	**Prognostic significance***
KIT mutations	t(8;21)(q22;q22.1)	Disease-free [18], relapse-free [19], event-free [19], [20], [21] and overall survival (OS) [18],[19], [20], [21],[22], [23] significantly shorter and cumulative incidence of relapse (CIR) [24] and relapse incidence (RI) [22] higher for patients with *KIT* mutations (especially those in exon 17) compared with patients with wild-type *KIT* No significant differences in complete remission (CR) rate [25], DFS [25], EFS [26], RR [25], [26]or OS [25], [26] between paediatric patients with and without *KIT* mutations in an American [25] and a Taiwanese [26] series. In a Japanese study, paediatric patients with *KIT* mutations (especially those in exon 17) had significantly

		shorter DFS and OS and higher risk of relapse (RR) than patients with wild-type *KIT* [27]
KIT mutations	inv(16)(p13.1q22)/ t(16;16)(p13.1;q22)	In most studies, there were no significant differences in RI [22], RFS [19], PFS [28], EFS [19] or OS [19], [22], [23], [28], [29] between patients with and without *KIT* mutations, or in EFS [21] or OS [21] between patients with and without *KIT* mutations in exon 17 at codon D816 Single studies reported shorter RFS [29] for patients with *KIT* mutations (especially those in exon 8), higher RR [30] for patients with exon 8 *KIT* mutations and higher CIR and shorter OS [24] for patients with exon 17 *KIT* mutations compared with patients with wild-type *KIT* In paediatric studies, no significant differences in (CR) rate [25], DFS [25], EFS [26], RR [25],[26] or OS [25],[26] between patients with and without *KIT* mutations were found
FLT3-ITD	Normal karyotype	DFS [31], [32], [33], CR duration (CRD) [34],[35] and OS [34],[35],[32],[33] significantly shorter for patients with *FLT3*-ITD compared with patients without *FLT3*-ITD CR rates not significantly different between patients with and without *FLT3*-ITD [31], [35], [32],[33]
FLT3-ITD with no expression of wild-type *FLT3*	Normal karyotype	DFS and OS significantly shorter for patients with *FLT3*-ITD and no expression of wild-type *FLT3* compared with patients without *FLT3*-ITD [31]
FLT3-ITD	Various abnormal and normal karyotypes combined	OS significantly shorter for younger (aged <60 y) patients with *FLT3*-ITD compared with patients without *FLT3*-ITD [23]
FLT3-ITD mutant level	Various abnormal and normal karyotypes combined	RR and OS (but not CR rate) increasingly bad for increasing *FLT3*-ITD mutant levels in a comparison of mutant levels in 4 subsets of patients: 1) without *FLT3*-ITD, 2) with low *FLT3*-ITD mutant level (i.e., when *FLT3*-ITD constituted 1%-24% of total *FLT3* alleles), 3) intermediate mutant level (25%-50%) and high mutant level (>50%) [36]
Biallelic *CEBPA* mutations	Normal karyotype	CR rates significantly higher [37] and DFS [38], RFS[37], EFS [37] and OS [38],[37], [39] significantly longer for patients with double *CEBPA* mutations compared with patients with wild-type *CEBPA* genes
Biallelic *CEBPA* mutations	Various abnormal and normal karyotypes combined	DFS [38], [40], EFS [41] and OS [38], [41], [42], [40] significantly longer for patients with double *CEBPA* mutations compared with patients with wild-type *CEBPA* genes and with patients with single *CEBPA* mutations.
Single *CEBPA* mutation	Normal karyotype	CR rates significantly lower [37] and DFS [38] and OS [38] shorter for patients with single *CEBPA* mutations compared with patients with double *CEBPA* mutations
NPM1 mutation	Normal karyotype	In some studies, patients with *NPM1* mutations had a significantly higher CR rate [43] and longer DFS [44], RFS [45] and EFS [43] than patients with wild-type *NPM1* genes, whereas in other studies, CR rates [46],[47], RFS [46], [43],[47] and EFS [46],[47] did not differ significantly between patients with and without an *NPM1* mutation. No significant differences in OS were consistently observed between patients with and without *NPM1* mutations

		[46], [43], [44], [45], [47] Older patients (aged ≥60 y) with an *NPM1* mutation had CR rate, DFS and OS significantly better than those of patients with wild-type *NPM1* genes [48]
NPM1 mutation & *FLT3*-ITD	Normal karyotype	CR rates [49], EFS [43], RFS [49] [45], DFS [44] and OS [49], [43], [44], [45] significantly better for patients with an *NPM1* mutation who lack *FLT3*-ITD compared with patients with an *NPM1* mutation and *FLT3*-ITD or those with wild-type *NPM1* genes with or without *FLT3*-ITD
RUNX1 mutation	Normal karyotype	CR rate significantly lower [50], resistant disease rate higher [51] and DFS [52], [50], EFS [53], [51], [50] and OS [52], [53], [50] shorter for patients with a *RUNX1* mutation compared with patients with wild-type *RUNX1* genes
RUNX1 mutation	Various abnormal and normal karyotypes combined	CR rates significantly lower [52], rates of resistant disease higher [51] and DFS [52], RFS [51], EFS [51] and OS [52], [51] shorter for patients with *RUNX1* mutations compared with patients with wild-type *RUNX1* genes
RUNX1 mutation	Non-complex karyotype (i.e., 1 or 2 abnormalities and a normal karyotype combined)	EFS and OS significantly shorter for patients with a *RUNX1* mutation compared with patients with wild-type *RUNX1* genes [53]
KMT2A-PTD	Normal karyotype	No differences in CR rates, DFS and OS between younger patients (aged <60 y) with and without *KMT2A*-PTD receiving intensive treatment that included autologous stem cell transplantation [54] or among older patients (aged ≥60 y) [55] In earlier studies, patients with *KMT2A*-PTD had significantly worse CRD (but not CR rate or OS) [56], [57] and higher risk of relapse or death during CR [49] than patients without *KMT2A*-PTD
KMT2A-PTD	Various abnormal and normal karyotypes combined	OS significantly shorter for younger (aged <60 y) patients with *KMT2A*-PTD compared with patients without *KMT2A*-PTD [23]
WT1 mutations	Normal karyotype	*WT1* mutations CR rates significantly lower [58], rates of resistant disease [58], RR [59] and CIR [58] higher and DFS [60], RFS [58], EFS [61] and OS [60] shorter for patients with *WT1* mutations compared with patients with wild-type *WT1* CR rates, RFS and OS significantly worse for younger (aged ≤60 y) patients with *WT1* mutations and *FLT3*-ITD compared with patients with mutated *WT1* without *FLT3*-ITD [62] CR rates [63], EFS [63], [64] and OS [63], [64] significantly worse for paediatric patients with a *WT1* mutation compared with patients with wild-type *WT1* genes in a European [63] and an American [64] study, whereas no significant differences in DFS or OS were found in a Japanese study [65]
WT1 mutations	Various abnormal and normal karyotypes combined	RR and OS significantly worse for patients with *WT1* mutations compared with patients with unmutated *WT1* [59]; EFS not significantly different [61] Rates of resistant disease [63], CIR [63], EFS [63], [64] and OS [63], [64] significantly worse for paediatric patients with a *WT1* mutation

		compared with patients with wild-type *WT1* in a European [63] and an American [64] study, whereas no significant differences in DFS or OS were found in a Japanese study [65]
TET2 mutations	Normal karyotype	No significant differences in CR rates [66,67,68], DFS [66], RFS[67,68], EFS [67,66] or OS [66,67,68, 69] between patients with *TET2* mutations and patients with wild-type *TET2* genes CR rates [66], DFS [66], RR [69], EFS [66,69]and OS [66] significantly worse for patients with *TET2* mutations classified in the ELN Favorable Genetic Group‡ (but not for those classified in the ELN Intermediate-I Group‡) compared with equivalent patients with wild-type *TET2*. One study [67] reported a higher CR rate for younger (aged ≤60 y) patients with a *TET2* mutation classified in the ELN Intermediate-I Genetic Group‡ (but not for those in the ELN Favourable Group‡) compared with patients with wild-type *TET2* RR [69], EFS [69] and OS [70], significantly worse for patients with a *TET2* mutation and an *NPM1* mutation without *FLT3*-ITD compared with patients with wild-type *TET2* genes. Similarly, *TET2* mutations were associated with shorter RFS and OS in younger patients (aged ≤60 y) with *FLT3*-ITD [68] and with shorter OS in *NPM1*-mutated patients [70]
TET2 mutation	Various abnormal and normal karyotypes combined	No significant differences in CR rates, RFS, EFS or OS between younger (aged ≤60 y) patients with *TET2* mutations and patients with wild-type *TET2* genes [67]
ASXL1 mutation	Normal karyotype	CR rates [71], DFS [71], EFS [72], [71] and OS [72], [71] significantly worse for patients with *ASXL1* mutations compared with patients with wild-type *ASXL1* genes CR rates, DFS, EFS and OS significantly worse for older (aged ≥60 y) patients with *ASXL1* mutations classified in the ELN Favourable Genetic Group‡ (but not for those classified in the ELN Intermediate-I Group‡) compared with the respective patients with wild-type *ASXL1* genes [71]
ASXL1 mutation	Various abnormal and normal karyotypes combined	CR rates [73],[74], [75], RFS [75] and OS [23],[73],[74],[75]significantly worse for patients with an *ASXL1* mutation than for patients with wild-type *ASXL1* genes.
ASXL1 mutation	Intermediate risk karyotype†	EFS and OS significantly shorter for patients with *ASXL1* mutations than for patients with wild-type *ASXL1* genes [72]
DNMT3A mutation	Normal karyotype	CR rates [76], DFS [77], EFS [78] and OS [78],[76] significantly worse for patients with a *DNMT3A* mutation (R882 and non-R882 mutations combined) than for patients with wild-type *DNMT3A* in some studies, but CR rates [79], RFS [79],[76], EFS [79]or OS [79] not different in other studies EFS and OS significantly shorter for younger *NPM1*-mutated patients (aged ≤60 y) with *DNMT3A* mutations (mostly R882) compared with patients with wild-type *DNMT3A* genes [78] RFS and OS significantly shorter for younger (aged ≤60 y) patients with *DNMT3A* mutations (mainly R882 mutations) classified in the ELN Intermediate-I Genetic Group‡ compared with patients with wild-type *DNMT3A* genes. No difference in outcome for patients with and without *DNMT3A* mutations

		classified in the ELN Favourable Genetic Group‡ [79] EFS and OS significantly shorter for younger (aged ≤60 y) patients who were *NPM1*-mutated/*FLT3*-ITD-negative or biallelic *CEBPA* mutation-positive [78]
R882 *DNMT3A* mutation	Normal karyotype	DFS and OS significantly shorter for older (≥60 y) patients with an R882 *DNMT3A* mutation compared with patients with wild-type *DNMT3A* genes [77]
non-R882 *DNMT3A* mutation	Normal karyotype	DFS significantly shorter for younger patients (aged <60 y) with *a* non-R882 *DNMT3A* mutation compared with patients with wild-type *DNMT3A* genes [77]
DNMT3A mutation	Various abnormal and normal karyotypes combined	CR rates significantly higher but RFS, EFS or OS not significantly different for younger patients (aged ≤60 y) with a *DNMT3A* mutation (R882 and non-R882 mutations combined) than for patients with wild-type *DNMT3A* [79]
IDH1 mutation	Normal karyotype	No significant differences in CR rate [80,81], DFS [80], RR [81] or OS [80,81] between patients with *IDH1* mutations and patients with wild-type *IDH1* and *IDH2* genes
R132 *IDH1* mutation	Normal karyotype	DFS significantly shorter for *NPM1*-mutated/*FLT3*-ITD-negative patients with an R132 *IDH1* mutation compared with patients with wild-type *IDH1* and *IDH2* genes [80] RR significantly higher and OS shorter for *NPM1*- or *CEBPA*-mutated/*FLT3*-ITD-negative patients with R132 *IDH1* mutations compared with patients with wild-type *IDH1* genes [81]
IDH2 mutation	Normal karyotype	No significant differences in CR rate, RFS or OS between patients with and without an *IDH2* mutation (mostly R140), which was also true in a subset of *NPM1*-mutated/*FLT3*-ITD-negative patients [82]
R172 *IDH2* mutation	Normal karyotype	CR rate [80,81], RR [81] and OS [81] significantly worse for patients with an R172 *IDH2* mutation compared with patients with wild-type *IDH2* genes
R140 *IDH2* mutation	Normal karyotype	No significant differences in CR rate, DFS or OS between patients with an R140 *IDH2* mutation and patients with wild-type *IDH1*and *IDH2* genes [80]
R140Q *IDH2* mutation	Various abnormal and normal karyotypes combined	OS significantly longer for younger (aged <60 y) patients with an R140Q *IDH2* mutation compared with patients with wild-type *IDH2* genes [23]
IDH1 and *IDH2* mutations combined	Normal karyotype	DFS and OS significantly shorter for patients with an *IDH1* or an *IDH2* mutation compared with patients with wild-type *IDH1* and *IDH2* genes [83] No significant differences in CR rate, RFS or OS between younger (aged ≤60 y) patients with *IDH1* or *IDH2* mutations compared with patients with wild-type *IDH1* and *IDH2* genes, but RFS significantly shorter in a subset of *NPM1*-mutated/*FLT3*-ITD-negative patients [84]
IDH1 and *IDH2* mutations combined	Various abnormal and normal karyotypes combined	No significant differences in CR rate, RFS or OS between younger (aged ≤60 y) patients with an *IDH1* mutation or with either an *IDH1* or an *IDH2* mutation compared with patients

		with wild-type *IDH1* and *IDH2* genes[84]
TP53 alterations (mutation or loss)	Complex karyotype (≥3 abnormalities)[l]	RFS, EFS and OS significantly shorter for patients with a *TP53* alteration compared with patients without a *TP53* alteration [85]
TP53 mutation	Complex karyotype (≥5 abnormalities)	No significant differences in CR rate, DFS or OS for patients with a *TP53* mutation compared with patients with wild-type *TP53* genes [86]
TP53 mutation	Abnormalities of chromosomes 5, 7 or 17 and/or complex karyotype (≥5 abnormalities)	OS significantly shorter for patients with *TP53* mutations compared with patients with wild-type *TP53* genes [86]
BAALC expression	Normal karyotype	CR rates [87,88], rates of primary resistant disease [87], DFS [32,89,88], EFS [89], RR [87], CIR [87] and OS [32,87,89,88] significantly worse for patients with high expression of the *BAALC* gene in blood compared with patients with low expression of the *BAALC* gene No significant differences in CIR or EFS between paediatric patients with high and low *BAALC* expression, whereas OS was significantly shorter in univariate but not in multivariate analysis [90]
BAALC expression	Various abnormal and normal karyotypes combined	No significant differences in CIR [90], EFS [90] or OS [90,91] between paediatric patients with high and low *BAALC* expression. In one study [91], high *BAALC* expression was associated with a significantly shorter EFS
ERG expression	Normal karyotype	CR rate [92,93], DFS [93], EFS [92], CIR [94], and OS [88,94,93] significantly worse for patients with high *ERG* expression in blood [94,92] or in bone marrow [93] compared with patients with low *ERG* expression No significant differences in CIR, EFS or OS between paediatric patients with high and low *ERG* expression [90]
ERG expression	Various abnormal and normal karyotypes combined	No significant differences in CIR, EFS or OS between paediatric patients with high *ERG* expression and patients with low *ERG* expression [90]
MN1 expression	Normal karyotype	CR rate significantly lower [95,96], RR higher [97] and RFS [97], EFS [96] and OS [97,95] shorter for patients with high *MN1* expression compared with patients with low *MN1* expression
DNMT3B expression	Normal karyotype	CR rate significantly lower and DFS and OS shorter for older (aged ≥60 y) patients with high *DNMT3B* expression compared with patients with low *DNMT3B* expression [98]
SPARC expression	Normal karyotype	CR rate significantly lower and DFS and OS shorter for younger (aged <60 y) patients with high *SPARC* expression compared with patients with low *SPARC* expression [99]
MECOM/EVI1 expression	Normal karyotype	EFS significantly shorter for younger patients (aged ≤60 y) with high *MECOM/EVI1* expression compared with patients with low *MECOM/EVI1* expression [100]
MECOM/EVI1 expression	Various abnormal and normal karyotypes combined	CR rate significantly lower and RFS and EFS shorter for younger patients (aged ≤60 y) with high *MECOM/EVI1* expression compared with patients with low *MECOM/EVI1* expression [100]

MECOM/EVI1 expression	Intermediate risk karyotype†	RFS and EFS significantly shorter for younger patients (aged ≤60 y) with high *MECOM/EVI1* expression compared with patients with low *MECOM/EVI1* expression [100]
miR-181a expression	Normal karyotype	CR rates and OS significantly better for younger (aged <60 y) patients with high *miR-181a* expression compared with patients with low *miR-181a* expression [101]
miR-3151 expression	Normal karyotype	DFS and OS significantly shorter for older patients (aged ≥60 y) with high *miR-3151* expression compared with patients with low *miR-3151* expression [102]
miR-3151 expression	Intermediate risk karyotype†	DFS and OS significantly shorter and CIR higher for patients with high *miR-3151* expression compared with patients with low *miR-3151* expression [103]
miR-155 expression	Normal karyotype	CR rates significantly lower and DFS and OS shorter for patients with high *miR-155* expression compared with patients with low *miR-155* expression [104]

CIR, cumulative incidence of relapse; CR, complete remission; CRD, complete remission duration; CIR, DFS, disease-free survival; EFS, event-free survival; ELN, European LeukemiaNet; *FLT3*-ITD, internal tandem duplication of the *FLT3* gene; *FLT3*-TKD, mutations in the tyrosine kinase domain of the *FLT3* gene; *KMT2A*-PTD, partial tandem duplication of the *KMT2A* (*MLL*) gene; NA, not applicable ; OS, overall survival; PFS, progression-free survival; RFS, relapse-free survival; RI, relapse incidence; RR, risk of relapse; y, years.

* Data presented pertain to adult patients, unless otherwise indicated.

† According to the refined Medical Research Council criteria[105].

‡ Cytogenetically normal patients classified in the ELN Favourable Genetic Group have mutated *CEBPA* and/or mutated *NPM1* without *FLT3*-ITD, whereas patients classified in the ELN Intermediate-I Genetic Group have wild-type *CEBPA* genes and either wild-type *NPM1* with or without *FLT3*-ITD or mutated *NPM1* with *FLT3*-ITD.

§ This group includes patients with wild-type *NPM1* genes with or without *FLT3*-ITD, or with mutated *NPM1* with *FLT3*-ITD.

‖ Complex karyotype defined by ELN as ≥3 chromosome abnormalities in the absence of the WHO-designated recurring translocations or inversions, i.e., t(8;21), inv(16) or t(16;16), t(15;17), t(9;11), t(v;11)(v;q23.3), t(6;9), inv(3) or t(3;3).

Supplemental References:

1. Ma Z, Morris SW, Valentine V, et al. Fusion of two novel genes, RBM15 and MKL1, in the t(1;22)(p13;q13) of acute megakaryoblastic leukemia. *Nat Genet*. 2001;28(3):220-221.
2. Mercher T, Coniat MB, Monni R, et al. Involvement of a human gene related to the Drosophila spen gene in the recurrent t(1;22) translocation of acute megakaryocytic leukemia. *Proc Natl Acad Sci USA*. 2001;98(10):5776-5779.
3. Beverloo HB, Panagopoulos I, Isaksson M, et al. Fusion of the homeobox gene HLXB9 and the ETV6 gene in infant acute myeloid leukemias with the t(7;12)(q36;p13). *Cancer Res*. 2001;61(14):5374-5377.
4. Harrison CJ, Hills RK, Moorman AV, et al. Cytogenetics of childhood acute myeloid leukemia: United Kingdom Medical Research Council Treatment trials AML 10 and 12. *J Clin Oncol*. 2010;28(16):2674-2681.

5. von Bergh AR, van Drunen E, van Wering ER, et al. High incidence of t(7;12)(q36;p13) in infant AML but not in infant ALL, with a dismal outcome and ectopic expression of HLXB9. *Genes Chromosomes Cancer*. 2006;45(8):731-739.
6. Coenen EA, Zwaan CM, Reinhardt D, et al. Pediatric acute myeloid leukemia with t(8;16)(p11;p13), a distinct clinical and biological entity: a collaborative study by the International-Berlin-Frankfurt-Munster AML-study group. *Blood*. 2013;122(15):2704-2713.
7. Sandahl JD, Coenen EA, Forestier E, et al. t(6;9)(p22;q34)/DEK-NUP214-rearranged pediatric myeloid leukemia: an international study of 62 patients. *Haematologica*. 2014;99(5):865-872.
8. Tarlock K, Alonzo TA, Moraleda PP, et al. Acute myeloid leukaemia (AML) with t(6;9)(p23;q34) is associated with poor outcome in childhood AML regardless of FLT3-ITD status: a report from the Children's Oncology Group. *Br J Haematol*. 2014;166(2):254-259.
9. Felix CA, Lange BJ. Leukemia in infants. *Oncologist*. 1999;4(3):225-240.
10. Krauter J, Wagner K, Schafer I, et al. Prognostic factors in adult patients up to 60 years old with acute myeloid leukemia and translocations of chromosome band 11q23: individual patient data-based meta-analysis of the German Acute Myeloid Leukemia Intergroup. *J Clin Oncol*. 2009;27(18):3000-3006.
11. Balgobind BV, Raimondi SC, Harbott J, et al. Novel prognostic subgroups in childhood 11q23/MLL-rearranged acute myeloid leukemia: results of an international retrospective study. *Blood*. 2009;114(12):2489-2496.
12. Ostronoff F, Othus M, Gerbing RB, et al. NUP98/NSD1 and FLT3/ITD coexpression is more prevalent in younger AML patients and leads to induction failure: a COG and SWOG report. *Blood*. 2014;124(15):2400-2407.
13. Hollink IH, van den Heuvel-Eibrink MM, Arentsen-Peters ST, et al. NUP98/NSD1 characterizes a novel poor prognostic group in acute myeloid leukemia with a distinct HOX gene expression pattern. *Blood*. 2011;118(13):3645-3656.
14. de Rooij JD, Hollink IH, Arentsen-Peters ST, et al. NUP98/JARID1A is a novel recurrent abnormality in pediatric acute megakaryoblastic leukemia with a distinct HOX gene expression pattern. *Leukemia*. 2013;27(12):2280-2288.
15. Gruber TA, Larson Gedman A, Zhang J, et al. An Inv(16)(p13.3q24.3)-encoded CBFA2T3-GLIS2 fusion protein defines an aggressive subtype of pediatric acute megakaryoblastic leukemia. *Cancer Cell*. 2012;22(5):683-697.
16. Masetti R, Pigazzi M, Togni M, et al. CBFA2T3-GLIS2 fusion transcript is a novel common feature in pediatric, cytogenetically normal AML, not restricted to FAB M7 subtype. *Blood*. 2013;121(17):3469-3472.
17. Tarlock K, Meshinchi S. Pediatric acute myeloid leukemia: biology and therapeutic implications of genomic variants. *Pediatr Clin North Am*. 2015;62(1):75-93.
18. Park SH, Lee HJ, Kim IS, et al. Incidences and Prognostic Impact of c-KIT, WT1, CEBPA, and CBL Mutations, and Mutations Associated With Epigenetic Modification in Core Binding Factor Acute Myeloid Leukemia: A Multicenter Study in a Korean Population. *Ann Lab Med*. 2015;35(3):288-297.
19. Boissel N, Leroy H, Brethon B, et al. Incidence and prognostic impact of c-Kit, FLT3, and Ras gene mutations in core binding factor acute myeloid leukemia (CBF-AML). *Leukemia*. 2006;20(6):965-970.
20. Schnittger S, Kohl TM, Haferlach T, et al. KIT-D816 mutations in AML1-ETO-positive AML are associated with impaired event-free and overall survival. *Blood*. 2006;107(5):1791-1799.
21. Kim HJ, Ahn HK, Jung CW, et al. KIT D816 mutation associates with adverse outcomes in core binding factor acute myeloid leukemia, especially in the subgroup with RUNX1/RUNX1T1 rearrangement. *Ann Hematol*. 2013;92(2):163-171.
22. Cairoli R, Beghini A, Grillo G, et al. Prognostic impact of c-KIT mutations in core binding factor leukemias: an Italian retrospective study. *Blood*. 2006;107(9):3463-3468.

23. Patel JP, Gonen M, Figueroa ME, et al. Prognostic relevance of integrated genetic profiling in acute myeloid leukemia. *The New England journal of medicine*. 2012;366(12):1079-1089.
24. Paschka P, Marcucci G, Ruppert AS, et al. Adverse prognostic significance of KIT mutations in adult acute myeloid leukemia with inv(16) and t(8;21): a Cancer and Leukemia Group B Study. *J Clin Oncol*. 2006;24(24):3904-3911.
25. Pollard JA, Alonzo TA, Gerbing RB, et al. Prevalence and prognostic significance of KIT mutations in pediatric patients with core binding factor AML enrolled on serial pediatric cooperative trials for de novo AML. *Blood*. 2010;115(12):2372-2379.
26. Shih LY, Liang DC, Huang CF, et al. Cooperating mutations of receptor tyrosine kinases and Ras genes in childhood core-binding factor acute myeloid leukemia and a comparative analysis on paired diagnosis and relapse samples. *Leukemia*. 2008;22(2):303-307.
27. Shimada A, Taki T, Tabuchi K, et al. KIT mutations, and not FLT3 internal tandem duplication, are strongly associated with a poor prognosis in pediatric acute myeloid leukemia with t(8;21): a study of the Japanese Childhood AML Cooperative Study Group. *Blood*. 2006;107(5):1806-1809.
28. Jones D, Yao H, Romans A, et al. Modeling interactions between leukemia-specific chromosomal changes, somatic mutations, and gene expression patterns during progression of core-binding factor leukemias. *Genes Chromosomes Cancer*. 2010;49(2):182-191.
29. Paschka P, Du J, Schlenk RF, et al. Secondary genetic lesions in acute myeloid leukemia with inv(16) or t(16;16): a study of the German-Austrian AML Study Group (AMLSG). *Blood*. 2013;121(1):170-177.
30. Care RS, Valk PJ, Goodeve AC, et al. Incidence and prognosis of c-KIT and FLT3 mutations in core binding factor (CBF) acute myeloid leukaemias. *Br J Haematol*. 2003;121(5):775-777.
31. Whitman SP, Archer KJ, Feng L, et al. Absence of the wild-type allele predicts poor prognosis in adult de novo acute myeloid leukemia with normal cytogenetics and the internal tandem duplication of FLT3: a cancer and leukemia group B study. *Cancer Res*. 2001;61(19):7233-7239.
32. Bienz M, Ludwig M, Leibundgut EO, et al. Risk assessment in patients with acute myeloid leukemia and a normal karyotype. *Clin Cancer Res*. 2005;11(4):1416-1424.
33. Whitman SP, Maharry K, Radmacher MD, et al. FLT3 internal tandem duplication associates with adverse outcome and gene- and microRNA-expression signatures in patients 60 years of age or older with primary cytogenetically normal acute myeloid leukemia: a Cancer and Leukemia Group B study. *Blood*. 2010;116(18):3622-3626.
34. Frohling S, Schlenk RF, Breitruck J, et al. Prognostic significance of activating FLT3 mutations in younger adults (16 to 60 years) with acute myeloid leukemia and normal cytogenetics: a study of the AML Study Group Ulm. *Blood*. 2002;100(13):4372-4380.
35. Beran M, Luthra R, Kantarjian H, Estey E. FLT3 mutation and response to intensive chemotherapy in young adult and elderly patients with normal karyotype. *Leuk Res*. 2004;28(6):547-550.
36. Gale RE, Green C, Allen C, et al. The impact of FLT3 internal tandem duplication mutant level, number, size, and interaction with NPM1 mutations in a large cohort of young adult patients with acute myeloid leukemia. *Blood*. 2008;111(5):2776-2784.
37. Taskesen E, Bullinger L, Corbacioglu A, et al. Prognostic impact, concurrent genetic mutations, and gene expression features of AML with CEBPA mutations in a cohort of 1182 cytogenetically normal AML patients: further evidence for CEBPA double mutant AML as a distinctive disease entity. *Blood*. 2011;117(8):2469-2475.
38. Hou HA, Lin LI, Chen CY, Tien HF. Reply to 'Heterogeneity within AML with CEBPA mutations; only CEBPA double mutations, but not single CEBPA mutations are associated with favorable prognosis'. *Br J Cancer*. 2009;101(4):738-740.

39. Dufour A, Schneider F, Metzeler KH, et al. Acute myeloid leukemia with biallelic CEBPA gene mutations and normal karyotype represents a distinct genetic entity associated with a favorable clinical outcome. *J Clin Oncol*. 2010;28(4):570-577.
40. Pabst T, Eyholzer M, Fos J, Mueller BU. Heterogeneity within AML with CEBPA mutations; only CEBPA double mutations, but not single CEBPA mutations are associated with favourable prognosis. *Br J Cancer*. 2009;100(8):1343-1346.
41. Wouters BJ, Lowenberg B, Erpelinck-Verschueren CA, van Putten WL, Valk PJ, Delwel R. Double CEBPA mutations, but not single CEBPA mutations, define a subgroup of acute myeloid leukemia with a distinctive gene expression profile that is uniquely associated with a favorable outcome. *Blood*. 2009;113(13):3088-3091.
42. Green CL, Koo KK, Hills RK, Burnett AK, Linch DC, Gale RE. Prognostic significance of CEBPA mutations in a large cohort of younger adult patients with acute myeloid leukemia: impact of double CEBPA mutations and the interaction with FLT3 and NPM1 mutations. *J Clin Oncol*. 2010;28(16):2739-2747.
43. Schnittger S, Schoch C, Kern W, et al. Nucleophosmin gene mutations are predictors of favorable prognosis in acute myelogenous leukemia with a normal karyotype. *Blood*. 2005;106(12):3733-3739.
44. Thiede C, Koch S, Creutzig E, et al. Prevalence and prognostic impact of NPM1 mutations in 1485 adult patients with acute myeloid leukemia (AML). *Blood*. 2006;107(10):4011-4020.
45. Dohner K, Schlenk RF, Habdank M, et al. Mutant nucleophosmin (NPM1) predicts favorable prognosis in younger adults with acute myeloid leukemia and normal cytogenetics: interaction with other gene mutations. *Blood*. 2005;106(12):3740-3746.
46. Boissel N, Renneville A, Biggio V, et al. Prevalence, clinical profile, and prognosis of NPM mutations in AML with normal karyotype. *Blood*. 2005;106(10):3618-3620.
47. Bardet V, Costa LD, Elie C, et al. Nucleophosmin status may influence the therapeutic decision in de novo acute myeloid leukemia with normal karyotype. *Leukemia*. 2006;20(9):1644-1646.
48. Becker H, Marcucci G, Maharry K, et al. Favorable prognostic impact of NPM1 mutations in older patients with cytogenetically normal de novo acute myeloid leukemia and associated gene- and microRNA-expression signatures: a Cancer and Leukemia Group B study. *J Clin Oncol*. 2010;28(4):596-604.
49. Schlenk RF, Dohner K, Krauter J, et al. Mutations and treatment outcome in cytogenetically normal acute myeloid leukemia. *N Engl J Med*. 2008;358(18):1909-1918.
50. Mendler JH, Maharry K, Radmacher MD, et al. RUNX1 mutations are associated with poor outcome in younger and older patients with cytogenetically normal acute myeloid leukemia and with distinct gene and MicroRNA expression signatures. *J Clin Oncol*. 2012;30(25):3109-3118.
51. Gaidzik VI, Bullinger L, Schlenk RF, et al. RUNX1 mutations in acute myeloid leukemia: results from a comprehensive genetic and clinical analysis from the AML study group. *J Clin Oncol*. 2011;29(10):1364-1372.
52. Tang JL, Hou HA, Chen CY, et al. AML1/RUNX1 mutations in 470 adult patients with de novo acute myeloid leukemia: prognostic implication and interaction with other gene alterations. *Blood*. 2009;114(26):5352-5361.
53. Schnittger S, Dicker F, Kern W, et al. RUNX1 mutations are frequent in de novo AML with noncomplex karyotype and confer an unfavorable prognosis. *Blood*. 2011;117(8):2348-2357.
54. Whitman SP, Ruppert AS, Marcucci G, et al. Long-term disease-free survivors with cytogenetically normal acute myeloid leukemia and MLL partial tandem duplication: a Cancer and Leukemia Group B study. *Blood*. 2007;109(12):5164-5167.
55. Whitman SP, Caligiuri MA, Maharry K, et al. The MLL partial tandem duplication in adults aged 60 years and older with de novo cytogenetically normal acute myeloid leukemia. *Leukemia*. 2012;26(7):1713-1717.

56. Caligiuri MA, Strout MP, Lawrence D, et al. Rearrangement of ALL1 (MLL) in acute myeloid leukemia with normal cytogenetics. *Cancer Res*. 1998;58(1):55-59.
57. Dohner K, Tobis K, Ulrich R, et al. Prognostic significance of partial tandem duplications of the MLL gene in adult patients 16 to 60 years old with acute myeloid leukemia and normal cytogenetics: a study of the Acute Myeloid Leukemia Study Group Ulm. *J Clin Oncol*. 2002;20(15):3254-3261.
58. Virappane P, Gale R, Hills R, et al. Mutation of the Wilms' tumor 1 gene is a poor prognostic factor associated with chemotherapy resistance in normal karyotype acute myeloid leukemia: the United Kingdom Medical Research Council Adult Leukaemia Working Party. *J Clin Oncol*. 2008;26(33):5429-5435.
59. Renneville A, Boissel N, Zurawski V, et al. Wilms tumor 1 gene mutations are associated with a higher risk of recurrence in young adults with acute myeloid leukemia: a study from the Acute Leukemia French Association. *Cancer*. 2009;115(16):3719-3727.
60. Paschka P, Marcucci G, Ruppert AS, et al. Wilms' tumor 1 gene mutations independently predict poor outcome in adults with cytogenetically normal acute myeloid leukemia: a cancer and leukemia group B study. *J Clin Oncol*. 2008;26(28):4595-4602.
61. Krauth MT, Alpermann T, Bacher U, et al. WT1 mutations are secondary events in AML, show varying frequencies and impact on prognosis between genetic subgroups. *Leukemia*. 2015;29(3):660-667.
62. Gaidzik VI, Schlenk RF, Moschny S, et al. Prognostic impact of WT1 mutations in cytogenetically normal acute myeloid leukemia: a study of the German-Austrian AML Study Group. *Blood*. 2009;113(19):4505-4511.
63. Hollink IH, van den Heuvel-Eibrink MM, Zimmermann M, et al. Clinical relevance of Wilms tumor 1 gene mutations in childhood acute myeloid leukemia. *Blood*. 2009;113(23):5951-5960.
64. Ho PA, Zeng R, Alonzo TA, et al. Prevalence and prognostic implications of WT1 mutations in pediatric acute myeloid leukemia (AML): a report from the Children's Oncology Group. *Blood*. 2010;116(5):702-710.
65. Sano H, Shimada A, Tabuchi K, et al. WT1 mutation in pediatric patients with acute myeloid leukemia: a report from the Japanese Childhood AML Cooperative Study Group. *Int J Hematol*. 2013;98(4):437-445.
66. Metzeler KH, Maharry K, Radmacher MD, et al. TET2 mutations improve the new European LeukemiaNet risk classification of acute myeloid leukemia: a Cancer and Leukemia Group B study. *J Clin Oncol*. 2011;29(10):1373-1381.
67. Gaidzik VI, Paschka P, Spath D, et al. TET2 Mutations in Acute Myeloid Leukemia (AML): Results From a Comprehensive Genetic and Clinical Analysis of the AML Study Group. *J Clin Oncol*. 2012;30(12):1350-1357.
68. Damm F, Markus B, Thol F, et al. TET2 mutations in cytogenetically normal acute myeloid leukemia: clinical implications and evolutionary patterns. *Genes Chromosomes Cancer*. 2014;53(10):824-832.
69. Weissmann S, Alpermann T, Grossmann V, et al. Landscape of TET2 mutations in acute myeloid leukemia. *Leukemia*. 2012;26(5):934-942.
70. Tian X, Xu Y, Yin J, et al. TET2 gene mutation is unfavorable prognostic factor in cytogenetically normal acute myeloid leukemia patients with NPM1+ and FLT3-ITD - mutations. *Int J Hematol*. 2014;100(1):96-104.
71. Metzeler KH, Becker H, Maharry K, et al. ASXL1 mutations identify a high-risk subgroup of older patients with primary cytogenetically normal AML within the ELN Favorable genetic category. *Blood*. 2011;118(26):6920-6929.
72. Schnittger S, Eder C, Jeromin S, et al. ASXL1 exon 12 mutations are frequent in AML with intermediate risk karyotype and are independently associated with an adverse outcome. *Leukemia*. 2013;27(1):82-91.

73. Chou WC, Huang HH, Hou HA, et al. Distinct clinical and biological features of de novo acute myeloid leukemia with additional sex comb-like 1 (ASXL1) mutations. *Blood*. 2010;116(20):4086-4094.
74. Pratcorona M, Abbas S, Sanders MA, et al. Acquired mutations in ASXL1 in acute myeloid leukemia: prevalence and prognostic value. *Haematologica*. 2012;97(3):388-392.
75. Paschka P, Schlenk RF, Gaidzik VI, et al. ASXL1 mutations in younger adult patients with acute myeloid leukemia: a study by the German-Austrian Acute Myeloid Leukemia Study Group. *Haematologica*. 2015;100(3):324-330.
76. Thol F, Damm F, Ludeking A, et al. Incidence and Prognostic Influence of DNMT3A Mutations in Acute Myeloid Leukemia. *J Clin Oncol*. 2011;29(21):2889-2896.
77. Marcucci G, Metzeler KH, Schwind S, et al. Age-related prognostic impact of different types of DNMT3A mutations in adults with primary cytogenetically normal acute myeloid leukemia. *J Clin Oncol*. 2012;30(7):742-750.
78. 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.
79. Gaidzik VI, Schlenk RF, Paschka P, et al. Clinical impact of DNMT3A mutations in younger adult patients with acute myeloid leukemia: results of the AML Study Group (AMLSG). *Blood*. 2013;121(23):4769-4777.
80. Marcucci G, Maharry K, Wu YZ, et al. IDH1 and IDH2 gene mutations identify novel molecular subsets within de novo cytogenetically normal acute myeloid leukemia: a Cancer and Leukemia Group B study. *J Clin Oncol*. 2010;28(14):2348-2355.
81. Boissel N, Nibourel O, Renneville A, et al. Prognostic impact of isocitrate dehydrogenase enzyme isoforms 1 and 2 mutations in acute myeloid leukemia: a study by the Acute Leukemia French Association group. *J Clin Oncol*. 2010;28(23):3717-3723.
82. Thol F, Damm F, Wagner K, et al. Prognostic impact of IDH2 mutations in cytogenetically normal acute myeloid leukemia. *Blood*. 2010;116(4):614-616.
83. Nomdedeu J, Hoyos M, Carricondo M, et al. Adverse impact of IDH1 and IDH2 mutations in primary AML: experience of the Spanish CETLAM group. *Leuk Res*. 2012;36(8):990-997.
84. Paschka P, Schlenk RF, Gaidzik VI, et al. IDH1 and IDH2 mutations are frequent genetic alterations in acute myeloid leukemia and confer adverse prognosis in cytogenetically normal acute myeloid leukemia with NPM1 mutation without FLT3 internal tandem duplication. *J Clin Oncol*. 2010;28(22):3636-3643.
85. Rucker FG, Schlenk RF, Bullinger L, et al. TP53 alterations in acute myeloid leukemia with complex karyotype correlate with specific copy number alterations, monosomal karyotype, and dismal outcome. *Blood*. 2012;119(9):2114-2121.
86. Bowen D, Groves MJ, Burnett AK, et al. TP53 gene mutation is frequent in patients with acute myeloid leukemia and complex karyotype, and is associated with very poor prognosis. *Leukemia*. 2009;23(1):203-206.
87. Baldus CD, Thiede C, Soucek S, Bloomfield CD, Thiel E, Ehninger G. BAALC expression and FLT3 internal tandem duplication mutations in acute myeloid leukemia patients with normal cytogenetics: prognostic implications. *J Clin Oncol*. 2006;24(5):790-797.
88. Schwind S, Marcucci G, Maharry K, et al. BAALC and ERG expression levels are associated with outcome and distinct gene and microRNA expression profiles in older patients with de novo cytogenetically normal acute myeloid leukemia: a Cancer and Leukemia Group B study. *Blood*. 2010;116(25):5660-5669.
89. Baldus CD, Tanner SM, Ruppert AS, et al. BAALC expression predicts clinical outcome of de novo acute myeloid leukemia patients with normal cytogenetics: a Cancer and Leukemia Group B Study. *Blood*. 2003;102(5):1613-1618.
90. Hermkens MC, van den Heuvel-Eibrink MM, Arentsen-Peters ST, et al. The clinical relevance of BAALC and ERG expression levels in pediatric AML. *Leukemia*. 2013;27(3):735-737.

91. Staffas A, Kanduri M, Hovland R, et al. Presence of FLT3-ITD and high BAALC expression are independent prognostic markers in childhood acute myeloid leukemia. *Blood*. 2011;118(22):5905-5913.
92. Marcucci G, Maharry K, Whitman SP, et al. High expression levels of the ETS-related gene, ERG, predict adverse outcome and improve molecular risk-based classification of cytogenetically normal acute myeloid leukemia: a Cancer and Leukemia Group B Study. *J Clin Oncol*. 2007;25(22):3337-3343.
93. Metzeler KH, Dufour A, Benthaus T, et al. ERG expression is an independent prognostic factor and allows refined risk stratification in cytogenetically normal acute myeloid leukemia: a comprehensive analysis of ERG, MN1, and BAALC transcript levels using oligonucleotide microarrays. *J Clin Oncol*. 2009;27(30):5031-5038.
94. Marcucci G, Baldus CD, Ruppert AS, et al. Overexpression of the ETS-related gene, ERG, predicts a worse outcome in acute myeloid leukemia with normal karyotype: a Cancer and Leukemia Group B study. *J Clin Oncol*. 2005;23(36):9234-9242.
95. Langer C, Marcucci G, Holland KB, et al. Prognostic importance of MN1 transcript levels, and biologic insights from MN1-associated gene and microRNA expression signatures in cytogenetically normal acute myeloid leukemia: a cancer and leukemia group B study. *J Clin Oncol*. 2009;27(19):3198-3204.
96. Schwind S, Marcucci G, Kohlschmidt J, et al. Low expression of MN1 associates with better treatment response in older patients with de novo cytogenetically normal acute myeloid leukemia. *Blood*. 2011;118(15):4188-4198.
97. Heuser M, Beutel G, Krauter J, et al. High meningioma 1 (MN1) expression as a predictor for poor outcome in acute myeloid leukemia with normal cytogenetics. *Blood*. 2006;108(12):3898-3905.
98. Niederwieser C, Kohlschmidt J, Volinia S, et al. Prognostic and biologic significance of DNMT3B expression in older patients with cytogenetically normal primary acute myeloid leukemia. *Leukemia*. 2015;29(3):567-575.
99. Alachkar H, Santhanam R, Maharry K, et al. SPARC promotes leukemic cell growth and predicts acute myeloid leukemia outcome. *J Clin Invest*. 2014;124(4):1512-1524.
100. Groschel S, Lugthart S, Schlenk RF, et al. High EVI1 expression predicts outcome in younger adult patients with acute myeloid leukemia and is associated with distinct cytogenetic abnormalities. *J Clin Oncol*. 2010;28(12):2101-2107.
101. Schwind S, Maharry K, Radmacher MD, et al. Prognostic significance of expression of a single microRNA, miR-181a, in cytogenetically normal acute myeloid leukemia: a Cancer and Leukemia Group B study. *J Clin Oncol*. 2010;28(36):5257-5264.
102. Eisfeld AK, Marcucci G, Maharry K, et al. miR-3151 interplays with its host gene BAALC and independently affects outcome of patients with cytogenetically normal acute myeloid leukemia. *Blood*. 2012;120(2):249-258.
103. Diaz-Beya M, Brunet S, Nomdedeu J, et al. The expression level of BAALC-associated microRNA miR-3151 is an independent prognostic factor in younger patients with cytogenetic intermediate-risk acute myeloid leukemia. *Blood Cancer J*. 2015;5:e352.
104. Marcucci G, Maharry KS, Metzeler KH, et al. Clinical role of microRNAs in cytogenetically normal acute myeloid leukemia: miR-155 upregulation independently identifies high-risk patients. *J Clin Oncol*. 2013;31(17):2086-2093.
105. Grimwade D, Hills RK, Moorman AV, et al. Refinement of cytogenetic classification in acute myeloid leukemia: determination of prognostic significance of rare recurring chromosomal abnormalities among 5876 younger adult patients treated in the United Kingdom Medical Research Council trials. *Blood*. 2010;116(3):354-365.

Appendix

Chairs and members of the Clinical Advisory Committee for myeloid neoplasms

Clinicians:
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