**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).

<table>
<thead>
<tr>
<th>Translocation</th>
<th>Gene Fusions</th>
<th>Frequency in Children/Frequency in Adults</th>
<th>Age Group Predilection</th>
<th>Comments/Prognosis</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>t(1;22)(p13.3;q13.1)</td>
<td>RBM15(OTT)-MKL1(MAL)</td>
<td>0.8%/0%</td>
<td>Infants</td>
<td>AMKL – FAB M7/Intermediate</td>
<td>1, 2</td>
</tr>
<tr>
<td>t(7;12)(q36.3;p13.2)</td>
<td>MNX1-ETV6</td>
<td>0.8%/&lt;0.5%</td>
<td>Infants</td>
<td>+19 seen as secondary abnormality Adverse</td>
<td>3, 4, 5</td>
</tr>
<tr>
<td>t(8;16)(p11.2;p13.3)</td>
<td>KAT6A-CREBBP</td>
<td>0.5%/&lt;0.5%</td>
<td>Infants and children</td>
<td>Can spontaneously remit in infancy; intermediate prognosis in later childhood</td>
<td>6</td>
</tr>
<tr>
<td>t(6;9)(p23;q34.1)</td>
<td>DEK-NUP214</td>
<td>1.7%/1%</td>
<td>Older children; rare in infants</td>
<td>Adverse; 65% with FLT3-ITD</td>
<td>7, 8</td>
</tr>
<tr>
<td>11q23.3</td>
<td>KMT2A (MLL) translocated</td>
<td>25%/5-10%</td>
<td>Infant 50%</td>
<td>Prognosis dependent on the partner gene</td>
<td>9, 10</td>
</tr>
<tr>
<td>t(9;11)(p21.3;q23.3)</td>
<td>KMT2A-MLLT3</td>
<td>9.5%/2%</td>
<td>Children</td>
<td>Intermediate</td>
<td>11</td>
</tr>
<tr>
<td>Molecular Genetic Alteration</td>
<td>Cytogenetic group</td>
<td>Prognostic significance*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-------------------</td>
<td>--------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>t(10;11)(p12;q23.3)</td>
<td>KMT2A-MLLT10</td>
<td>3.5%/1% Children</td>
<td>Include subtle and cryptic KMT2A rearrangements/Adverse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>t(6;11)(q27;q23.3)</td>
<td>KMT2A-MLLT4</td>
<td>2%/&lt;0.5% Children</td>
<td>Adverse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>t(1;11)(q21;q23.3)</td>
<td>KMT2A-MLLT11</td>
<td>1%/&lt;0.5% Children</td>
<td>Favorable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>t(5;11)(q35.3;p15.5)</td>
<td>NUP98-NSD1</td>
<td>7%/3% 16% of FLT3-ITD patients</td>
<td>Adverse; 80% with FLT3-ITD. In combination associated with induction failure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>inv(16)(p13.3q24.3)</td>
<td>CBFA2T3-GLIS2</td>
<td>3%/0% 10% of Infants, 20% of FAB M7</td>
<td>Adverse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>t(11;12)(p15.5;p13.5)</td>
<td>NUP98-KDM5A</td>
<td>3%/0% Children &lt;5 years 10% of FAB M7</td>
<td>Intermediate</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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

KIT mutations: t(8;21)(q22;q22.1)

- Disease-free, relapse-free, event-free, and overall survival (OS) significantly shorter and cumulative incidence of relapse (CIR) and relapse incidence (RI) 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, DFS, EFS, RR or OS between paediatric patients with and without KIT mutations in an American and a Taiwanese 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*.

### KIT Mutations

| Mutation Type | Karyotype | Outcome
|---------------|-----------|--------|
| *KIT* inv(16)(p13.1q22)/t(16;16)(p13.1;q22) | Normal karyotype | In most studies, there were no significant differences in RI, RFS, EFS or OS between patients with and without *KIT* mutations, or in EFS or OS between patients with and without *KIT* mutations in exon 17 at codon D816. Single studies reported shorter RFS for patients with *KIT* mutations (especially those in exon 8), higher RR for patients with exon 8 *KIT* mutations and higher CIR and shorter OS for patients with exon 17 *KIT* mutations compared with patients with wild-type *KIT*. In paediatric studies, no significant differences in (CR) rate, DFS, EFS, RR or OS between patients with and without *KIT* mutations were found.

### FLT3-ITD

| Mutation Type | Karyotype | Outcome
|---------------|-----------|--------|
| Normal karyotype | DFS, CR duration, OS 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.

### FLT3-ITD with no expression of wild-type FLT3

| Karyotype | Outcome
|-----------|--------|
| Various abnormal and normal karyotypes combined | OS significantly shorter for younger (aged <60 y) patients with FLT3-ITD compared with patients without FLT3-ITD.

### FLT3-ITD Mutant Level

| Karyotype | Outcome
|-----------|--------|
| 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%).

### Biallelic CEBPA Mutations

| Karyotype | Outcome
|-----------|--------|
| Normal karyotype | CR rates significantly higher and DFS, RFS, EFS significantly longer for patients with double CEBPA mutations compared with patients with wild-type CEBPA genes.

### Biallelic CEBPA Mutations

| Karyotype | Outcome
|-----------|--------|
| Various abnormal and normal karyotypes combined | DFS, OS 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

| Karyotype | Outcome
|-----------|--------|
| Normal karyotype | CR rates significantly lower and DFS and OS shorter for patients with single CEBPA mutations compared with patients with double CEBPA mutations.

### NPM1 Mutation

| Karyotype | Outcome
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal karyotype</td>
<td>In some studies, patients with NPM1 mutations had a significantly higher CR rate and longer DFS, RFS and EFS than patients with wild-type NPM1 genes, whereas in other studies, CR rates, RFS and EFS 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.</td>
</tr>
</tbody>
</table>
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.

<table>
<thead>
<tr>
<th>Mutation Combination</th>
<th>Karyotype Description</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>NPM1 mutation &amp; FLT3-ITD</td>
<td>Normal karyotype</td>
<td>CR rates significantly better for patients with NPM1 mutation who lack FLT3-ITD compared with patients with NPM1 mutation and FLT3-ITD or those with wild-type NPM1 genes with or without FLT3-ITD</td>
</tr>
<tr>
<td>RUNXI mutation</td>
<td>Normal karyotype</td>
<td>CR rate significantly lower, resistant disease rate higher, and DFS, EFS, RFS, and OS shorter for patients with RUNXI mutations compared with patients with wild-type RUNXI genes</td>
</tr>
<tr>
<td>RUNXI mutation</td>
<td>Various abnormal and normal karyotypes combined</td>
<td>CR rates significantly lower, rates of resistant disease higher, and DFS, EFS, RFS, and OS shorter for patients with RUNXI mutations compared with patients with wild-type RUNXI genes</td>
</tr>
<tr>
<td>RUNXI mutation</td>
<td>Non-complex karyotype (i.e., 1 or 2 abnormalities and a normal karyotype combined)</td>
<td>EFS and OS significantly shorter for patients with RUNXI mutation compared with patients with wild-type RUNXI genes</td>
</tr>
<tr>
<td>KMT2A-PTD</td>
<td>Normal karyotype</td>
<td>No differences in CR rates, DFS and OS between younger patients (aged &lt;60 y) with and without KMT2A-PTD receiving intensive treatment that included autologous stem cell transplantation or among older patients (aged ≥60 y)</td>
</tr>
<tr>
<td>KMT2A-PTD</td>
<td>Various abnormal and normal karyotypes combined</td>
<td>OS significantly shorter for younger (aged &lt;60 y) patients with KMT2A-PTD compared with patients without KMT2A-PTD</td>
</tr>
<tr>
<td>WT1 mutations</td>
<td>Normal karyotype</td>
<td>WT1 mutations CR rates significantly lower, rates of resistant disease, RR, and CIR higher and DFS, RFS, EFS, and OS shorter for patients with WT1 mutations compared with patients with wild-type WT1</td>
</tr>
<tr>
<td>WT1 mutations</td>
<td>Various abnormal and normal karyotypes combined</td>
<td>RR and OS significantly worse for patients with WT1 mutations compared with patients with unmutated WT1; EFS not significantly different</td>
</tr>
<tr>
<td>WT1 mutations</td>
<td>Various abnormal and normal karyotypes combined</td>
<td>Rates of resistant disease, CIR, EFS, and OS significantly worse for paediatric patients with a WT1 mutation</td>
</tr>
</tbody>
</table>
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 \(^65\).

<p>| <strong>TET2 mutations</strong> | Normal karyotype | No significant differences in CR rates (^66), DFS (^66), RR (^69), EFS (^66), (^67), (^68), (^69), OS (^66), (^67), (^68), (^69) between patients with <em>TET2</em> mutations and patients with wild-type <em>TET2</em> genes. CR rates (^66), DFS (^66), RR (^69), EFS (^66), (^67), (^68), (^69), and OS (^66) significantly worse for patients with <em>TET2</em> 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 <em>TET2</em>. One study (^67) reported a higher CR rate for younger (aged ≤60 y) patients with a <em>TET2</em> mutation classified in the ELN Intermediate-I Genetic Group (but not for those in the ELN Favourable Group) compared with patients with wild-type <em>TET2</em>. RR (^69), EFS (^69) and OS (^70), significantly worse for patients with a <em>TET2</em> mutation and an <em>NPM1</em> mutation without FLT3-ITD compared with patients with wild-type <em>TET2</em> genes. Similarly, <em>TET2</em> mutations were associated with shorter RFS and OS in younger patients (aged ≤60 y) with FLT3-ITD (^68) and with shorter OS in <em>NPM1</em>-mutated patients (^70). |
| <strong>TET2 mutation</strong> | Various abnormal and normal karyotypes combined | No significant differences in CR rates, RFS, EFS or OS between younger (aged ≤60 y) patients with <em>TET2</em> mutations and patients with wild-type <em>TET2</em> genes (^67). |
| <strong>ASXL1 mutation</strong> | Normal karyotype | CR rates (^71), DFS (^71), EFS (^71), (^72) and OS (^72), (^71) significantly worse for patients with <em>ASXL1</em> mutations compared with patients with wild-type <em>ASXL1</em> genes. CR rates, DFS, EFS and OS significantly worse for older (aged ≥60 y) patients with <em>ASXL1</em> mutations classified in the ELN Favourable Genetic Group (but not for those classified in the ELN Intermediate-I Genetic Group) compared with the respective patients with wild-type <em>ASXL1</em> genes (^71). |
| <strong>ASXL1 mutation</strong> | Various abnormal and normal karyotypes combined | CR rates (^71), (^74), (^75), RFS (^75) and OS (^73), (^74), (^75), (^72) significantly worse for patients with an <em>ASXL1</em> mutation than for patients with wild-type <em>ASXL1</em> genes. |
| <strong>ASXL1 mutation</strong> | Intermediate risk karyotype† | EFS and OS significantly shorter for patients with <em>ASXL1</em> mutations than for patients with wild-type <em>ASXL1</em> genes (^72). |
| <strong>DNMT3A mutation</strong> | Normal karyotype | CR rates (^76), DFS (^77), EFS (^78) and OS (^78), (^76) significantly worse for patients with a <em>DNMT3A</em> mutation (R882 and non-R882 mutations combined) than for patients with wild-type <em>DNMT3A</em> 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 <em>NPM1</em>-mutated patients (aged ≤60 y) with <em>DNMT3A</em> mutations (mostly R882) compared with patients with wild-type <em>DNMT3A</em> genes (^78). RFS and OS significantly shorter for younger (aged ≤60 y) patients with <em>DNMT3A</em> mutations (mainly R882 mutations) classified in the ELN Intermediate-I Genetic Group (but not for those in the ELN Favourable Group) compared with patients with wild-type <em>DNMT3A</em> genes. No difference in outcome for patients with and without <em>DNMT3A</em> mutations. |</p>
<table>
<thead>
<tr>
<th>Mutation</th>
<th>Karyotype</th>
<th>Description</th>
</tr>
</thead>
</table>
| **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  
EFS and OS significantly shorter for younger (aged ≤60 y) patients who were NPM1-mutated/FLT3-ITD-negative or biallelic CEBPA mutation-positive |
| **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  
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)  
less than for patients with wild-type DNMT3A  
and wild-type NPM1 and CEBPA genes  
No significant differences in CR rate, RFS, EFS 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  
No significant differences in CR rate, DFS 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  
No significant differences in CR rate, DFS 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  
No significant differences in CR rate, DFS 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  
No significant differences in CR rate, DFS 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 |
| **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)  
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  
3 vs 4.5 months for patients with and without an IDH2 mutation (mostly R140), and 3 vs 4.5 months for patients with wild-type IDH2 genes |
| **IDH1 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  
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  
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  
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  
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 |
| **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  
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  
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  
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  
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 |
| **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  
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  
3 vs 4.5 months for patients with and without an IDH2 mutation (mostly R140), and 3 vs 4.5 months for patients with wild-type IDH2 genes |
| **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  
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  
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  
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  
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 |
| **R172 IDH2 mutation**                        | Normal karyotype                   | CR rate 6.5 vs 8 months for patients with and without an IDH2 mutation (mostly R140), and 6.5 vs 8 months for patients with wild-type IDH2 genes  
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  
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  
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  
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  
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 |
| **R140 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  
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  
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  
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  
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 |
<p>| <strong>R140Q IDH2 mutation</strong>                       | Various abnormal and normal karyotypes combined | OS significantly longer for younger (aged &lt;60 y) patients with an R140Q IDH2 mutation compared with patients with wild-type IDH2 genes |
| <strong>IDH1 and IDH2 mutations combined</strong>          | 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 |
| <strong>IDH1 and IDH2 mutations combined</strong>          | 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 |</p>
<table>
<thead>
<tr>
<th>Gene Expression</th>
<th>Karyotypic Abnormalities</th>
<th>Clinical Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>TP53 alterations (mutation or loss)</td>
<td>Complex karyotype (≥3 abnormalities)</td>
<td>RFS, EFS and OS significantly shorter for patients with a TP53 alteration compared with patients without a TP53 alteration.</td>
</tr>
<tr>
<td>TP53 mutation</td>
<td>Complex karyotype (≥5 abnormalities)</td>
<td>No significant differences in CR rate, DFS or OS for patients with a TP53 mutation compared with patients with wild-type TP53 genes.</td>
</tr>
<tr>
<td>TP53 mutation</td>
<td>Abnormalities of chromosomes 5, 7 or 17 and/or complex karyotype (≥5 abnormalities)</td>
<td>OS significantly shorter for patients with TP53 mutations compared with patients with wild-type TP53 genes.</td>
</tr>
<tr>
<td>BAALC expression</td>
<td>Normal karyotype</td>
<td>CR rates, rates of primary resistant disease, DFS, EFS, RR, CIR, and OS significantly worse for patients with high expression of the BAALC gene in blood compared with patients with low expression of the BAALC gene.</td>
</tr>
<tr>
<td>BAALC expression</td>
<td>Various abnormal and normal karyotypes combined</td>
<td>No significant differences in CIR, EFS or OS between paediatric patients with high and low BAALC expression.</td>
</tr>
<tr>
<td>ERG expression</td>
<td>Normal karyotype</td>
<td>CR rate, DFS, EFS, and OS significantly worse for patients with high ERG expression in blood or in bone marrow compared with patients with low ERG expression.</td>
</tr>
<tr>
<td>ERG expression</td>
<td>Various abnormal and normal karyotypes combined</td>
<td>No significant differences in CIR, EFS or OS between paediatric patients with high and low ERG expression.</td>
</tr>
<tr>
<td>MN1 expression</td>
<td>Normal karyotype</td>
<td>CR rate significantly lower and DFS and OS shorter for older (aged ≥60 y) patients with high MN1 expression compared with patients with low MN1 expression.</td>
</tr>
<tr>
<td>DNMT3B expression</td>
<td>Normal karyotype</td>
<td>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.</td>
</tr>
<tr>
<td>SPARC expression</td>
<td>Normal karyotype</td>
<td>CR rate significantly lower and DFS and OS shorter for younger (aged &lt;60 y) patients with high SPARC expression compared with patients with low SPARC expression.</td>
</tr>
<tr>
<td>MECOM/EVI1 expression</td>
<td>Normal karyotype</td>
<td>EFS significantly shorter for younger patients (aged ≤60 y) with high MECOM/EVI1 expression compared with patients with low MECOM/EVI1 expression.</td>
</tr>
<tr>
<td>MECOM/EVI1 expression</td>
<td>Various abnormal and normal karyotypes combined</td>
<td>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.</td>
</tr>
<tr>
<td>Expression/Expression</td>
<td>Karyotype</td>
<td>#<strong>RFS and EFS significantly shorter for younger patients (aged ≤60 y) with high MECOM/EVI1 expression compared with patients with low MECOM/EVI1 expression</strong></td>
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<tr>
<td><strong>MECOM/EVI1</strong></td>
<td>Intermediate risk karyotype(^{†})</td>
<td></td>
</tr>
<tr>
<td><strong>miR-181a</strong></td>
<td>Normal karyotype</td>
<td><strong>RFS and EFS significantly shorter for younger patients (aged ≤60 y) with high miR-181a expression compared with patients with low miR-181a expression</strong></td>
</tr>
<tr>
<td><strong>miR-3151</strong></td>
<td>Normal karyotype</td>
<td><strong>DFS and OS significantly shorter for older patients (aged ≥60 y) with high miR-3151 expression compared with patients with low miR-3151 expression</strong></td>
</tr>
<tr>
<td><strong>miR-3151</strong></td>
<td>Intermediate risk karyotype(^{†})</td>
<td></td>
</tr>
<tr>
<td><strong>miR-155</strong></td>
<td>Normal karyotype</td>
<td><strong>DFS and OS significantly shorter and CIR higher for patients with high miR-3151 expression compared with patients with low miR-3151 expression</strong></td>
</tr>
</tbody>
</table>

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.
\(^{ǁ}\) 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(11;v)(v;q23.3), t(6;9), inv(3) or t(3;3).

Supplemental References:


Appendix

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