

Table 17.3 Cytogenetics for favorable, intermediate, and poor prognosis for AML (*Continued*)

- ^aGregory TK, Wald D, Chen Y, Vermaat JM, Xiong Y, Tse W. Molecular prognostic markers for adult acute myeloid leukemia with normal cytogenetics. *J Hematol Oncol.* 2009;2:23.
- ^bBAALC is located on chromosome 8 at 8q22.3. BAALC has GenBank Accession No. AF363578.
- ^cERG is located on chromosome 21, at 21q22.3. ERG has GenBank Accession No. NM_004449.
- ^dHerry A, Douet-Guilbert N, Morel F, Le Bris MJ, De Braekeleer M. Redefining monosomy 5 by molecular cytogenetics in 23 patients with MDS/AML. *Eur. J. Haematol.* 2007; 78:457–467.
- ^eBram S, Swolin B, Rödger S, Stockelberg D, Ogård I, Bäck H. Is monosomy 5 an uncommon aberration? Fluorescence in situ hybridization reveals translocations and deletions in myelodysplastic syndromes or acute myelocytic leukemia. *Cancer Genet Cytogenet.* 2003;142:107–114.
- ^fHasle H, Alonzo TA, Auvrignon A, et al. Monosomy 7 and deletion 7q in children and adolescents with acute myeloid leukemia: an international retrospective study. *Blood.* 2007;109:4641–4647.
- ^gHarrison 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;28:2674–2681.
- ^hTamai H, Yamaguchi H, Hamaguchi H, et al. Clinical features of adult acute leukemia with 11q23 abnormalities in Japan: a co-operative multicenter study. *Int J Hematol.* 2008;87(2):195–202.
- ⁱZangrando A, Dell'orto MC, Te Kronnie G, Basso G. MLL rearrangements in pediatric acute lymphoblastic and myeloblastic leukemias: MLL specific and lineage specific signatures. *BMC Med Genomics.* 2009;2:36 (12 pages).
- ^jMarcucci G, Mrózek K, Ruppert AS, et al. Prognostic factors and outcome of core binding factor acute myeloid leukemia patients with t(8;21) differ from those of patients with inv(16): a Cancer and Leukemia Group B study. *J Clin Oncol.* 2005;23:5705–5717.
- ^kPayton JE, Grieselhuber NR, Chang LW, et al. High throughput digital quantification of mRNA abundance in primary human acute myeloid leukemia samples. *J Clin Invest.* 2009;119:1714–1726.
- ^lPetrie K, Zelent A. AML1/ETO, a promiscuous fusion oncoprotein. *Blood.* 2007;109:4109–4110.
- ^mXiao Z, Greaves MF, Buzflor P, et al. Molecular characterization of genomic AML1-ETO fusions in childhood leukemia. *Leukemia.* 2001;15:1906–1913.

1. Numeric abnormalities in ALL

Patients with hyperdiploid ALL, in particular those with more than 50 chromosomes, have the best prognosis. Hypodiploidy (less than 44 chromosomes), which is found in less than 2% of pediatric or adult cases, predicts a poor outcome. The rare cases with low hypodiploidy (33 to 39 chromosomes) and near-haploidy (23 to 29 chromosomes) have a particularly poor prognosis.

2. Structural abnormality t(9;22) (Philadelphia chromosome) in ALL

The outcome of ALL patients with blasts containing the Philadelphia chromosome is poor (171). The Philadelphia chromosome in ALL may be different from that found in chronic myeloid leukemia (CML). In ALL, this chromosome involves band 34 of the long arm of chromosome 9, splicing the proto-oncogene *c-abl* to band 11 of the long arm of chromosome 22 in the *bcr* gene. In 50 to 80% of cases of ALL, the breakpoint in 22q11 falls between exons b1 and b2 of the major breakpoint cluster region, as opposed to between b2 and b3 or b3 and b4 in chronic myeloid leukemia. The difference is in the positions of the breakpoints occurring in the translocation, that is, breakpoints within the BCR gene. This difference in breakpoints results in a smaller

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