## +11 or trisomy 11 (solely)

François Desangles  
Laboratoire de Biologie, Hôpital du Val de Grâce, 75230 Paris, France

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### Clinics and pathology

**Disease**  
Myeloid lineage: (ANLL, MDS)

**Phenotype / cell stem origin**  
M1, M2, and M4 ANLL; therapy related ANLL; MDS evolving towards ANLL; stem cell immunophenotype (DR+, CD34+, and CD15, 33 and/or 13 positive); trilineage dysplasia may be present.  
To be noted that M1 and M2 subtypes of ANLL have rarely been found associated with the classical MLL rearrangements.

**Epidemiology**  
Frequency: 1% of ANLL and MDS as well; balanced sex ratio; found in adults; med age: 60 yrs.

**Prognosis**  
Short CR; poor prognosis.

### Cytogenetics

**Cytogenetics, morphological**

+11

**Cytogenetics, molecular**  
Partial tandem duplication (in situ) of MLL gene located in 11q23.

**Probes**  
Oncor, Inc.

**Additional anomalies**  
None (by that very fact).

### Genes involved and Proteins

**MLL**  
Location: 11q23  
DNA / RNA  
21 exons, spanning over 100 kb; 13-15 kb mRNA.

**Protein**  
431 kDa; contains two DNA binding motifs (a AT hook, and Zinc fingers), a DNA methyl transferase motif, a bromodomain; wide expression; nuclear localisation; transcriptional regulatory factor.

### Results of the chromosomal anomaly

**Hybrid gene**  
**Description**  
Exons 1 to 6 or 8 fused to a nearly entire MLL gene, starting at exon 2 (i.e. the duplicated segment is E2 to E6 or 8).

**Fusion protein**  
**Description**  
AT hook and DNA methyltransferase from MLL in N-term fused to a quite entire MLL in C-term.

**Expression localisation**  
Nuclear localisation.

**Oncogenesis**  
Probable altered transcriptional regulation.

**To be noted**  
Such a tandem duplication of MLL may also be found in cases with a normal karyotype.

### References


*This article should be referenced as such: Desangles F. +11 or trisomy 11 (solely). Atlas Genet Cytogenet Oncol Haematol.1997;1(1):12.*