Review on t(2;3)(p21;q26) THADA/MECOM, with data on clinics, and the genes implicated.

Identity

This translocation is found in a subset of cases described in the card t(2;3)(p15-23;q26-27). Other subsets involve other genes, such as BCL11A in the t(2;3)(p16;q26) BCL11A/MECOM.

Clinics and pathology

Disease

Acute myeloid leukemia (AML)

Epidemiology

One case to date, a 59-year old male patient with a M4-AML (Trubia et al., 2006).

Prognosis

Clinical outcome in cases with the t(2;3)(p16;q26) BCL11A/MECOM and the case with the t(2;3)(p21;q26) THADA/MECOM (plotted together) was severe: "One patient is alive with active disease at 12 months, five patients died after 4-14 months" (Trubia et al., 2006).

Genetics

MECOM was overexpressed.
Sequence specific DNA binding protein. Interacts with transcriptional coactivators, corepressors, and other sequence specific transcription factors. MECOM ("MDS1-EVI1") also contains a PR domain from "MDS1" in N-term (Wieser, 2008).

### Result of the chromosomal anomaly

#### Hybrid gene

**Description**

Regulatory sequences were transferred telomerically to MECOM.

#### Fusion protein

**Description**

The t(2;3) brings about the juxtaposition at 3q26 of the MECOM locus with regulatory elements normally located in proximity of the 2p breakpoints, with consequent EVI1 overexpression, without the formation of a fusion protein.

### References


Trubia M, Albano F, Cavazzini F, Cambrin GR et al.. Characterization of a recurrent translocation t(2;3)(p15-22;q26) occurring in acute myeloid leukaemia. Leukemia. 2006 Jan;20(1):48-54


This article should be referenced as such: