Leukaemia Section
Short Communication

t(10;10)(p12;q21) CTNNA3/ARHGAP21

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Abstract
Review on t(10;10)(p12;q21) , with data on clinics and the genes involved

Keywords
Chromosome 10; CTNNA3; ARHGAP21; Early T-cell precursor acute lymphoblastic leukaemia

Clinics and pathology

Disease
Early T-cell precursor acute lymphoblastic leukaemia

Epidemiology
Only one case to date, a 7-year old boy (Zhang et al. 2012).

Evolution
Outcome: relapse with lineage switch at 13 months after diagnosis; the patient died at 25 months post diagnosis.

Cytogenetics

Cytogenetics morphological
The karyotype was complex, with a constitutional inv(2)(p11.2q13), a del(1p), and other abnormalities.

Genes involved and proteins

To be noted, a missense mutation in DCLRE1C

ARHGAP21
Location 10p12.1
DNA/RNA

The transcript has 26 exons.

Protein
1958 amino acids (aa). ARHGAP21 acts as a Rho GTPase-activating protein for RHOA, RHOC and CDC42. ARHGAP21 plays a role in cell proliferation, migration, vesicle traffic, cell adhesions and insulin secretion. ARHGAP21 is implicated in many tumor tissues (Ferreira Pissarra et al. 2016).

CTNNA3

Location 10q21.3
DNA/RNA
The transcript has 18 exons.

Protein
895 aa for the longest isoform. CTNNA3 is an α-catenin. CTNNA3 plays a role in cell-cell adhesion: catenins link the single-pass type I transmembrane linkers proteins cadherins (cell-cell adhesion molecule expressed in adherents junctions) to the actin filament network. α-catenins bind the cytoplasmic domains of cadherins through β-catenin to actin filaments. An heterozygous constitutional (germline) deletion of CTNNA3 was found in a hybrid neurofibroma/schwannoma of a patient with clinically diagnosed neurofibromatosis type 2. Loss of CTNNA3 protein expression was found in neurofibromas, schwannomas, and malignant peripheral nerve sheath tumors. Depletion of CTNNA3 in Schwann cells was associated with reduced RNA of CDH1 (E-cadherin) via induction of SNAI1, SNAI2 (E-cadherin repressors) as well as disaggregation of the actin cytoskeleton and epithelial-mesenchymal transition promotion
CTNNA3, is a tumor suppressor in hepatocellular carcinoma, inhibiting the proliferation, migration and invasion. CTNNA3 inhibited AKT signal, and in turn decreased PCNA and MMP9, and increased the cell cycle inhibitor CDKN1A (p21Cip1/Waf1). CTNNA3 is also frequently mutated in laryngeal carcinomas and low-expressed in urothelial carcinoma of the bladder. Other α-catenins, CTNNA1 and CTNNA2 are also frequently mutated (loss of function) in various cancers
CTNNA3 is inhibited by MIR425 (He et al., 2016; Stahn et al., 2016).

### Result of the chromosomal anomaly

**Hybrid gene**
t(10;10)(p12;q21) CTNNA3/ARHGAP21 but no fusion transcript was detectable.

### References


