

# Leukaemia Section

## Short Communication

### t(6;10)(q22;q24) NFKB2/ROS1

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Printable original version : [http://documents.irevues.inist.fr/bitstream/handle/2042/70767/09-2019-t0610q22q24NFKB2\\_ROS1ID1720.pdf](http://documents.irevues.inist.fr/bitstream/handle/2042/70767/09-2019-t0610q22q24NFKB2_ROS1ID1720.pdf)  
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#### Abstract

Review on t(6;10)(q22;q24), with data on the genes involved.

#### Keywords

Chromosome 6; Chromosome 10; t(6;10)(q22;q24)

#### Clinics and pathology

##### Disease

Anaplastic large cell lymphoma, ALK negative

##### Note

Only one case to date (Crescenzo et al., 2015). No individual data, no clinical data.

##### Prognosis

No data

#### Genes involved and proteins

##### NFKB2

##### Location

10q24.32

##### Protein

The gene encodes a subunit of the transcription factor complex nuclear factor-kappa-B. NFKB2 is a rapidly acting primary transcription factor found in

all cell types. It is involved in the cellular responses to stimuli such as cytokines and stress and plays a key role in regulating the immunological response to infections.

##### ROS1

##### Location

6q22.1

##### Protein

This proto-oncogene belongs to the subfamily of tyrosine kinase insulin receptor genes and expresses in a variety of tumor cell lines.

ROS1 is an integral membrane protein with tyrosine kinase activity, that may function as a growth or differentiation factor receptor.

#### Result of the chromosomal anomaly

##### Hybrid gene

5' NFKB2 - 3' ROS1

##### Description

Nucleotide sequence analyses revealed that NFKB2 (exon 1-13) is fused to the intracytoplasmic domain of ROS1 accompanied by loss of the NFKB2 ankyrin region, that is necessary for the translation of constitutively active NFKB-fusion proteins and its oncogenic role [Crescenzo et al., 2015].



## Fusion protein

### Oncogenesis

NFKB2/ROS1 is a chimeric transcription factor with oncogenic potential that leads to the constitutive activation of STAT3 in the absence of either JAK-STAT3 mutations.

The ectopic expression of NFKB2/ROS1 is associated with the phosphorylation of JAK (JAK2 - JAK3) and STAT3. NFKB2/ROS1 fusion induces the expression of genes involved in membrane trafficking, replication, protein export, TNF signaling, T cell activation.

This, in turn, may lead to a widespread derangement of several metabolic and signaling pathways, which ultimately concur to neoplastic transformation [Crescenzo et al., 2015, Tabbo et al, 2016].

Crescenzo R, Abate F, Lasorsa E, Tabbo' F, Gaudiano M, Chiesa N, Di Giacomo F, Spaccarotella E, Barbarossa L, Ercole E, Todaro M, Boi M, Acquaviva A, Ficarra E, Novero D, Rinaldi A, Tousseyn T, Rosenwald A, Kenner L, Cerroni L, Tzankov A, Ponzoni M, Paulli M, Weisenburger D, Chan WC, Iqbal J, Piris MA, Zamo' A, Ciardullo C, Rossi D, Gaidano G, Pileri S, Tiacci E, Falini B, Shultz LD, Mevellec L, Vialard JE, Piva R, Bertoni F, Rabadan R, Inghirami G. Convergent mutations and kinase fusions lead to oncogenic STAT3 activation in anaplastic large cell lymphoma. *Cancer Cell*. 2015 Apr 13;27(4):516-32

Tabbò F, Pizzi M, Kyriakides PW, Ruggeri B, Inghirami G. Oncogenic kinase fusions: an evolving arena with innovative clinical opportunities. *Oncotarget*. 2016 May 3;7(18):25064-86

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## References