Leukaemia Section
Mini Review

\[ t(8;14)(q24;q11) \]

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\section*{Identity}

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\includegraphics[width=\textwidth]{figure1}
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\[ t(8;14)(q24;q11) \] G-banding - courtesy Charles Bangs and Leena Borkar, and R-banding (right) - courtesy Jacques Boyer.

\section*{Clinics and pathology}

\subsection*{Disease}
Acute lymphoblastic leukemia (ALL), (rare lymphoblastic lymphoma).

\subsection*{Phenotype/cell stem origin}
T lineage (TCR alpha beta + or TCR gamma delta). May be early-B lineage: conceivably the rare cases with early-B lineage represent a bipotential B-T lineage.

\subsection*{Epidemiology}
Rare: prevalence 0.5 to 1.3 % among all cases of ALL and about 2% among T ALL; male predominance; median age 5 years.

\subsection*{Clinics}
Bulky extramedullary leukemia, central nervous system (CNS) infiltration.

\subsection*{Cytology}
High white blood cell count.

\subsection*{Prognosis}
The disease progress rapidly and response to conventional therapy is poor, median survival 11 months.

\subsection*{Cytogenetics}

\subsection*{Additional anomalies}
Sole anomaly in half cases; additional anomalies are variable.
**Genes involved and proteins**

**Note**
On the molecular point of view the proto oncogen c-myc is juxtaposed with the gene of the T-cell receptor alpha chain (TCR-alpha).

**C-MYC**

**Location:** 8q24  
**DNA/RNA**
c-myc has three exons, two promoters P1 and P2 control the c-myc transcription.

**Protein**
Myc protein is a transcription factor of the helix loop leucin zipper family that activates transcription as obligate heterodimer with a partner protein, Max.

**TCR-alpha and TCR-delta**

**Location:** 14q11.2  
**DNA/RNA**
The TCR delta variable (V) diversity (D) joining (J) and constant region genes are situated within the TCR alpha locus between the TCR alpha V and the TCR alpha J segments. The TCR alpha/delta locus is transcribed in a centromer to telomer direction.

**Protein**
T-cell receptor.

**Result of the chromosomal anomaly**

**Hybrid gene**

**Description**
The majority of breakpoints are localised within the TCR delta gene although they may also involve TCR alpha J and rarely TCR alpha V. The 3’ part of the TCR alpha gene is relocated downstream of the c-myc protooncogene. The c-myc oncogene is not structurally altered.

**Transcript**
Detection of TCR alpha rearrangements is hampered by the large number of J segments.

**Fusion protein**

**Note**
No fusion protein but promoter exchange.

**Description**
High levels of electrophoretically normal p64 and p67 c-myc proteins are detected and both products keep their instability. Preferential utilization of P2 is maintained.

** Oncogenesis**
The activation of the gene myc is likely to result from its juxtaposition to the enhancer element of the TCR alpha gene with stimulates constitutive synthesis of normal c-myc.

**To be noted**

**Note**
The identification of a breakpoint to the 3’ side of c-myc suggest that this translocation is analogous to the t(2;8) and t(8;22) in Burkitt lymphoma. It is hypothesized that quantitative alteration of c-myc transcription alone may be sufficient for altered growth.

**References**


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