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Perspective

The Amplification of AML1 Variant in Cases of Acute Leukemia and its Role in Leukemogenesis

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DESCRIPTION

An essential transcription factor for the differentiation and proliferation of hematopoietic cells is *AML1/RUNX1*. By alternative splicing, the *AML1* gene can create at least three isoforms: AML1a, AML1b, and AML1c. AML1a prevents AML1b/1c, sometimes commonly referred to as *AML1*, from operating as intended. In the present investigation, the ALL patients expressed more AML1a than the controls did has been discovered.

Furthermore, AML1a counteracts the action of AML1b by repressing transcription from the promotor of the macrophage-colony stimulating factor receptor, which is mediated by AML1b. Bone Marrow Mononuclear Cells (BMMNCs) from mice were transduced with AML1a and transplanted into lethally irradiated animals, which develop lymphoblastic leukemia after transplantation, in order to study the role of AML1a in hematopoiesis and leukemogenesis *in vivo*.

AML1, a transcription factor also referred to as *RUNX1*, *PEBP2 αB* or *CBF α2* is essential for the growth and development of hematopoietic cells.

AML1 has two domains: a transactivation domain that binds to target genes and controls them, and a DNA binding domain called the Runt Homology Domain (RHD). Human leukemia is associated with chromosomal translocations t(8;21), t(3;21), and t(12;21) that influence *AML1*. The most common chromosomal translocation in acute myeloid leukemia is t(8;21), which connects *AML1* to ETO. This creates the *AML1/ETO* fusion gene, in which *AML1* has transactivation domain loss but RHD retention. Leukemogenesis is thought to be caused by the absence of trans activation domain. Hematopoiesis in both early development and adulthood depends on *AML1*. Because of a lack of fetal liver hematopoiesis and central nervous system bleeding, *AML1*-null embryos usually die at E12.5. Adult *AML1* deficiency causes several disruptions in hematopoiesis, such as delayed megakaryocytic maturation and compromised T and B-lymphocytic differentiation. Furthermore, it has been demonstrated that cells lacking *AML1* are vulnerable to malignant transformation.

The present investigation revealed that patients with acute lymphoblastic leukemia have an overexpression of AML1a. In contrast to essentially no expression of AML1a in normal persons, AML1a could be found in half of the AML patients using a qualitative assay for AML1a mRNA in a prior investigation. Here, the study extended to include additional patients using a semi-quantitative technique. Remarkably, were unable to detect any discernible variation in AML1a expression between the healthy donors and AML patients. Rather, a noteworthy distinction was discovered between the ALL patients and the control group. As a dominant negative protein, *AML1-ETO* prevents AML1b from transactivating the GM-CSF promoter. *AML1-ETO* is the initial hit, preventing Hematopoietic Stem Cell (HSC) development. When the second strike happens, myeloid leukemia develops. It was assumed that AML1a lacks transcriptional activity, just like *AML1-ETO*, but that it binds to target genes more firmly than AML1b, suggesting that it may play a role in leukemogenesis. Since it might affect myeloid differentiation and perhaps contribute to the development of leukemia, we have decided to investigate M-CSFR as the AML1 target gene. In our tests, AML1b trans activated the M-CSFR promoter activity in a dose-dependent manner, but AML1a did not. Wright-Giemsa staining solution was used to color PB smears and BM cytospin slides. Samples of tissue were embedded in paraffin and preserved with 10% phosphate-buffered formalin. Hematoxylin and Eosin (H&E) was used to stain the sections, which were then examined under a light microscope. The Kaplan-Meier estimates were used to create survival curves for statistical analysis. The student's t test was used to compare group distributions parametrically, and the Mann-Whitney U test or Chi-square test was used to compare group distributions non-parametrically.

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