

## **Molecular landscape of pediatric AML**

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Acute myeloid leukemia (AML) is the disease of elderly, where more than 80% of all cases of AML diagnosed in patients older than 60 years of age. Until recently, it was assumed that childhood AML is a less common variant of the adult disease. Recent studies into the underlying biology of childhood AML has defined the genomic and transcriptome of AML in younger patients and contrasted the underlying mechanism of disease to those in older adults. These studies have demonstrated that childhood AML is vastly different than the adult disease, with structural alterations including translocations/fusion transcripts leading to the generation of fusion oncoproteins are the predominant mechanism of disease in younger patients. As many of these disease defining fusions are associated with clinical outcome in children, comprehensive knowledge of such fusions, many of which are cryptic, can inform most appropriate risk and target based therapeutic interventions. Accurate identification of patients at high risk of relapse allows for allocation of patients to allogeneic stem cell transplantation in first CR prior to impending relapse. In this presentation we will review the most recent genomic and transcriptomic discoveries in childhood AML, their prognostic implications, therapeutic targets and most appropriate incorporation into the pediatric AML clinical trials.