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Exploring lncRNA-mRNA signatures in relapsed acute myeloid leukemia compared with primary tumors

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Motivation and Aim: Long non-coding RNAs (lncRNAs) play a role in the pathogenesis of acute myeloid leukemia (AML) by affecting gene expression, including in pediatric AML. Deregulation of lncRNAs patterns has the potential to regulate tumor drug resistance and sustaining of leukemic stem cells. Here we investigated the expression patterns of lncRNAs and mRNAs in relapsed tumors compared to primary tumors, using TARGET-AML project data.
Methods and Algorithms: We examined the molecular pathways for differentially expressed lncRNAs and mRNAs (using GO and LncPath) and constructed lncRNA-mRNA coexpression networks (Fig. 1, A, B for upregulated genes and Fig. 1, C, D for downregulated genes).

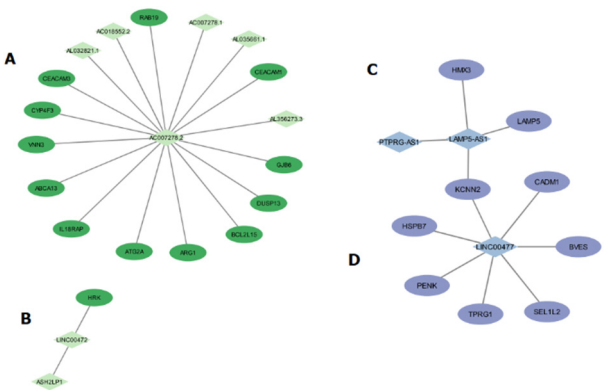

Fig. 1. lncRNA-mRNA co-expression networks of upregulated and downregulated genes. Upregulated mRNAs are shown as dark green ellipses; upregulated lncRNAs are shown as light green diamonds. Downregulated mRNAs are shown as lavender ellipses; downregulated lncRNAs are shown as light blue diamonds

Results: The lncRNA-mRNA landscape of relapsed tumors compared to primary tumors in pediatric AML is extremely inconsistent. In both upregulated networks and downregulated co-expression networks, there are parallel processes associated with the effects of therapy and mechanisms of drug resistance and immune system escape. Most of the genes involved in the interaction networks are nonspecific for a particular tumor type; intriguingly, some of them are associated with invasion processes that are not typical for hematological malignancies.
Conclusion: Detailed studies of the resulting interactions using other pediatric AML datasets are needed to better understanding the current results.