A new target in acute myeloid leukemia

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Acute myeloid leukemia, a common leukemia in adults, is characterized by aberrant proliferation of cancerous bone marrow cells. Activating mutations in a protein receptor known as FLT3 receptor are among the most prevalent mutations observed in acute myeloid leukemias. FLT3 mutants are thought to activate several signaling pathways that contribute to cancer development.

Dr. Daniel Tenen and colleagues from Harvard University in Boston discovered a new pathway activated by FLT3 mutation. Their results show that cyclin dependent kinase 1 (CDK1), a critical regulator of cell division is activated in FLT3 mutated leukemias, leading to the activation of downstream gene transcription.

Most importantly, they demonstrate that inhibiting CDK1 activity promotes differentiation of cells from patient-derived peripheral blood samples.

As clinical trials with CDK1 inhibitors are ongoing, their data strongly suggest that therapies targeting the CDK1 pathway may be efficacious for acute myeloid leukemias with FLT3 mutation, especially in patients resistant to FLT3 inhibitor therapies.

More information: Targeting CDK1 promotes FLT3-activated acute myeloid leukemia differentiation through C/EBPα, *Journal of Clinical Investigation*, 2012.