Study identifies leukemia tumor suppressor

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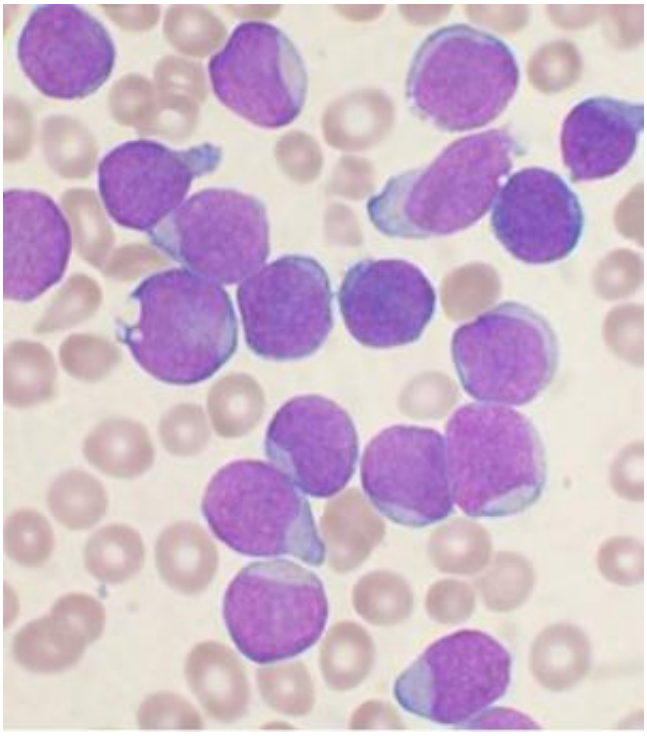

A Wright's stained bone marrow aspirate smear from a patient with precursor B-cell acute lymphoblastic leukemia. Credit: VashiDonsk/Wikipedia

A protein-coding gene called hnRNP K has been identified as a tumor suppressor for acute myeloid leukemia (AML), a finding that could be important for investigating how best to target treatment of a blood cancer striking mostly older individuals.

Expression of the protein-coding gene hnRNP K is significantly reduced in AML patients who carry a specific genetic deletion, according to a

study led by The University of Texas MD Anderson Cancer Center.

Study results were published in the Sept. 24 issue of *Cancer Cell*.

"Our data implicates hnRNP K in the development of blood disorders and suggests it acts as a tumor suppressor," said Sean Post, Ph.D., assistant professor of Leukemia. "Both in vivo and in vitro results indicate that hnRNP K achieves this through regulation of key genetic pathways. Our study found that hnRNP K expression must be maintained for proper cellular regulation and to prevent tumor formation."

Through use of a mouse model and cell lines, Post's team showed that reduced hnRNP K levels were associated with blood cancer. Their data revealed that AML patients who carried a partial deletion of chromosome 9 also experienced a significant decrease in hnRNP K expression. This deletion, 9q21.32, along with the decreased levels of hnRNP K, led to reduced survival and increased tumor formation.

HnRNP K was found to be haploinsufficient, in that it contained a single functional copy of a gene not capable of maintaining normal cellular function and leading to disease development.

"Our findings showed that hnRNP K haploinsufficiency led to tumor development by deregulating cell proliferation and differentiation programs through control of key cellular pathways, which suggests these pathways may be exploited by targeted therapies," said Post. "It was clear that these changes in AML patients with the 9q21.32 deletion resulted in a tumor suppressor gene involved in blood cancer development."

Provided by University of Texas M. D. Anderson Cancer Center