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Discovery of Protein Mutations may lead to Better Therapies for Leukemia

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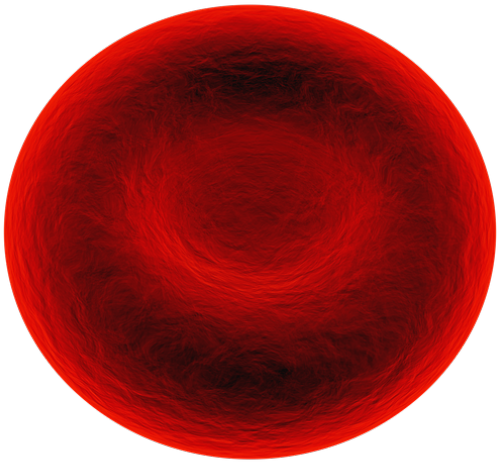
A team of scientists at Children's Hospital of Philadelphia (CHOP), led by Wei Tong, a hematology researcher reveals how mutated proteins cause several types of leukemia. This new understanding of the molecular mechanisms at work may offer clues to other drugs yet to be developed.



Researchers have uncovered how mutations in a protein network drive several high-risk leukemia, especially chronic myelomonocytic leukemia (CMML) and juvenile myelomonocytic leukemia (JMML), both of which tend to have a poor prognosis because they progress to acute myeloid leukemia (AML). This new understanding of the molecular mechanisms at work may offer clues to other drugs yet to be developed.

A team of scientists at Children's Hospital of Philadelphia (CHOP), led by Wei Tong, a hematology researcher reveals how mutated proteins cause several types of leukemia. According to a news source, the group investigated a well-known kinase, Janus kinase 2 (JAK2), which plays a key role in the development of blood-forming cells in bone marrow. If something disrupts the normal regulation of JAK2 activity, JAK2 triggers the uncontrolled growth of marrow cells that give rise to a myeloid leukemia. Until now, the molecular events that regulate JAK2 were poorly established.

Based on studies in animals and in primary human leukemia cells, Dr. Tong and colleagues report that mutations in either of two proteins, Casitas B-cell lymphoma (CBL) and LNK/SH2B3, form a complex with JAK2 to disrupt JAK2 regulation and cause leukemia.

Dr. Tong said to a news source that clinical research should test whether this drug can benefit patients with CMML and JMML, as well as AML patients who have CBL mutations.

In addition to the potential benefits of ruxolitinib, she continued, the team's findings may lead researchers to develop novel leukemia drugs aimed at mutations in any of the three proteins in a precision medicine approach.