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Robert H. Lurie Comprehensive Cancer Center of Northwestern University

Lurie Cancer Center's Basic Research Seminar Series

The Pathobiology of Myeloproliferative Neoplasms

Tuesday, May 7, 2024

11:00 a.m. - 12:00 p.m. CT

Baldwin Auditorium, 1st Floor
Robert H. Lurie Medical Research Center
303 E. Superior St., Chicago, IL

Host: Kehan Ren, PhD

Although the genes that drive the development of myeloid blood cancers have largely been defined, there are currently few effective targeted treatment strategies for these diseases, and none that are curative. This illuminates the need to exploit the molecular understanding that has been gained in the last decade through cancer exome sequencing to identify novel therapeutic vulnerabilities in myeloid malignancies. Research in the Elf Lab focuses on dissecting the molecular mechanisms underlying these diseases in order to identify unique molecular dependencies that can be targeted for therapeutic gain. MPNs are clonal hematopoietic disorders that arise from a single mutated hematopoietic stem cell and result in the multi-lineage expansion of mature myeloid cells. The classical MPNs comprise three distinct diseases: polycythemia vera (PV), essential thrombocythemia (ET), and myelofibrosis (MF). Nearly all MPNs are driven by somatic mutations in Janus kinase 2 (JAK2), calreticulin (CALR), or the thrombopoietin receptor (MPL). All three driver mutations are unified by their shared gain-of-function ability to constitutively activate JAK/STAT signaling. Accordingly, JAK inhibitors remain the only FDA-approved targeted therapy for the treatment of these diseases, and though they impart profound clinical benefit, they lack disease-modifying activity and are not curative.



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