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Identification and characterization of leukemia stem cells and phenotype-driving progenitors in myeloproliferative neoplasms

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Myeloproliferative neoplasms (MPNs) are a phenotypically diverse group of hematopoietic stem cell diseases characterized by overproduction of one or more of the myeloid cell lineages, currently in need of novel therapeutics. Unfortunately, the development of these agents are hindered by the lack of information on MPN initiating leukemia stem cells, and on the progenitors they produce to drive diverse MPN phenotypes. We have developed a unique set of three murine MPN models, which faithfully recapitulate MPN. Each model expresses a different gain-of-function mutation in the tyrosine kinase JAK2, resulting in a different MPN phenotype. Specifically, one results primarily in erythrocytosis, another in erythrocytosis and granulocytosis, and the third predominantly granulocytosis. In these models, we found a dramatic accumulation of a specific cell population in the bone marrow and spleen of diseased but not normal mice. This cell population expresses hematopoietic stem cells surface markers, is enriched in self-renewing activity, and causes MPN in secondary recipients. These results strongly suggest that this population represents MPN leukemia stem cells. In addition, we found two progenitor populations that are specifically expanded only in the two models with erythrocytosis, indicating that they are erythrocytosis-driving leukemic progenitors. We will discuss using animal models to characterize leukemia stem cells and MPN pathophysiology

Biography

Lily Huang has completed her Ph.D. from University of California at San Diego and postdoctoral studies from the Whitehead Institute for Biomedical Center at MIT. She has published more than 25 papers in reputed journals and serving as an editorial board member of Frontiers in Molecular and Structural Endocrinology.

Her current research interest is in the molecular mechanisms governing cytokine receptor signal transduction in hematopoietic stem and progenitor cells, and understanding how deregulation in these mechanisms results in hematological malignancies and cancer

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