RESEARCH ARTICLES

**Advances in the Treatment of Acute Myeloid Leukemia: New Drugs and New Challenges**

N.J. Short, M. Konopleva, T.M. Kadia, G. Barthakur, F. Ravandi, C.D. DiNardo, and N. Dave

**Characteristics and Outcome of AKT1E17K-Mutant Breast Cancer Defined through AACR Project GENIE, a Clinicogenomic Registry**


Précis: Data from AACR Project GENIE were used to investigate the effects of the rare AKT1E17K mutation in ER\(^+\) breast cancer, showing the challenges and advantages of using this type of real-world evidence.

See commentary, p. 490

**Monocytic Subclones Confer Resistance to Venetoclax-Based Therapy in Patients with Acute Myeloid Leukemia**


Précis: Compared with less differentiated acute myeloid leukemia (AML), monocytic AML is more resistant to venetoclax-based therapy, a phenomenon that may be attributable to dedifferentiation of preexisting monocytic subclones.

**LETTERS TO THE EDITOR**

Not Thoroughly Enough: Venetoclax-Amyloidosis Association

C. Zhu, A. Ushijima, S. Ahmed, G. Thomas, S. Xia, and K. Reinke

Précis: venetoclax therapy induced amyloidosis. The AML subpopulation of interest is unknown, and no specific guidelines to monitor patients for this adverse effect are available.

See commentary, p. 490
Impaired Death Receptor Signaling in Leukemia Causes Antigen-Independent Resistance by Inducing CAR T-cell Dysfunction ................. 552


Précis: Response to CD19-directed CAR T cells in acute lymphoblastic leukemia was dependent on death receptor signaling, and exposure to ALL cells with impaired death receptor signaling caused CAR T cells to adopt an exhausted-like phenotype.

See commentary, p. 492

Relapse-Fated Latent Diagnosis Subclones in Acute B Lineage Leukemia Are Drug Tolerant and Possess Distinct Metabolic Programs ................. 568


Précis: Minor subclones that are present at the time of diagnosis in B-progenitor acute lymphoblastic leukemia can cause relapse and exhibit distinct traits, including chemotherapy resistance, prior to treatment.

MYC Instructs and Maintains Pancreatic Adenocarcinoma Phenotype .......... 588


Précis: In a mouse model of mutant Kras-driven pancreatic ductal adenocarcinoma (PDAC), Myc activation was required for progression to PDAC, whereas Myc deactivation caused rapid regression of even established PDACs.

See commentary, p. 495

Oncogenic KRAS-Driven Metabolic Reprogramming in Pancreatic Cancer Cells Utilizes Cytokines from the Tumor Microenvironment .......... 608


Précis: In models of mutant Kras-driven pancreatic cancer, T H 2 cells in the tumor microenvironment produced the cytokines IL4 and IL13, which promoted metabolic reprogramming and tumorigenesis.