Dysregulated Hematopoiesis Caused by Mammary Cancer Is Associated with Epigenetic Changes and Hox Gene Expression in Hematopoietic Cells
Alexander Sio, Manreet K. Chehal, Kevin Tsai, Xueling Fan, Morgan E. Roberts, Brad H. Nelson, Jolanta Grembecka, Tomasz Cierpicki, Danielle L. Krebs, and Kenneth W. Harder

 précis: These findings provide insight into how tumor-secreted factors profoundly disturb hematopoiesis, for example by causing myeloproliferative-like disease (leukemoid reaction), anemia, and disrupted bone marrow stem compartments.

Adenomatous Polyps Are Driven by Microbe-Instigated Focal Inflammation and Are Controlled by IL-10–Producing T Cells
Kristen L. Dennis, Yunwei Wang, Nichole R. Blatner, Shuya Wang, Abdulrahman Saadalla, Erwin Truebo, Axel Roers, Casey T. Weaver, James J. Lee, Jack A. Gilbert, Eugene B. Chang, and Khashayarasha Khazaie

 précis: IL-10 provided by T cells in the colon is critical to control bacterial-driven inflammation and polyp growth, providing a rationale for this cytokine as a candidate target for immunotherapy in colon cancer.

Constitutive β-Catenin Activation Induces Male-Specific Tumorigenesis in the Bladder Urothelium
Congxing Lin, Yan Yin, Kristina Stemler, Peter Humphrey, Adam S. Kibel, Indira U. Mysorekar, and Liang Ma

 précis: Investigations in a preclinical model of bladder cancer suggest that males have a predilection for this disease due to a synergy between the β-catenin and androgen receptor signaling pathways.

FGFR4 Promotes Stroma-Induced Epithelial-to-Mesenchymal Transition in Colorectal Cancer
Rui Liu, Jingyi Li, Ke Xie, Tao Zhang, Yunlong Lei, Yi Chen, Lu Zhang, Kai Huang, Kui Wang, Hong Wu, Min Wu, Edouard C. Nice, Canhua Huang, and Yuquan Wei

 précis: An FGFR is found to be pivotal for the process by which the tumor stromal microenvironment triggers conversion of epithelial cancer cells to mesenchymal phenotypes that are more invasive and metastatic.
**PREVENTION AND EPIDEMIOLOGY**

**Chemoprevention of Prostate Cancer by D,L-Sulforaphane Is Augmented by Pharmacological Inhibition of Autophagy**
Avani R. Vyas, Eun-Ryeong Hahm, Julie A. Arlotti, Simon Watkins, Donna Beer Stoltz, Dhimant Desai, Shantu Amin, and Shivendra V. Singh

**Telomere Length in Peripheral Blood Lymphocytes Contributes to the Development of HPV-Associated Oropharyngeal Carcinoma**
Yang Zhang, Erich M. Sturgis, Kristina R. Dahlstrom, Juyi Wen, Hongliang Liu, Qingyi Wei, Guojun Li, and Zhensheng Liu

**THERAPEUTICS, TARGETS, AND CHEMICAL BIOLOGY**

**Indirubin Derivative 6BIO Suppresses Metastasis**
Simone Braig, Christine A. Kressirer, Johanna Liebl, Fabian Bischoff, Stefan Zahler, Laurent Meijer, and Angelika M. Vollmar

**Combination of Antibody That Inhibits Ligand-Independent HER3 Dimerization and a p110α Inhibitor Potently Blocks P13K Signaling and Growth of HER2+ Breast Cancers**

An Antibody That Locks HER3 in the Inactive Conformation Inhibits Tumor Growth Driven by HER2 or Neuregulin

Precis: HER3 is a member of the EGFR family that mediates oncogenic functions of other family members, thereby offering a target that can more generally shut down signaling by this common cancer cell system.

TUMOR AND STEM CELL BIOLOGY

Double Minute Chromosomes in Glioblastoma Multiforme Are Revealed by Precise Reconstruction of Oncogenic Amplicons
J. Zachary Sanborn, Sofie R. Salama, Mia Grifford, Cameron W. Brennan, Tom Mikkelsen, Suresh Jhanwar, Sol Katzman, Lynda Chin, and David Haussler

Precis: Oncogenic amplicons, a feature of many glioblastomas, were precisely reconstructed by high-throughout sequencing data, a process that could be useful for diagnosis and monitoring of disease.

MicroRNA-218 Inhibits Glioma Invasion, Migration, Proliferation, and Cancer Stem-like Cell Self-Renewal by Targeting the Polycomb Group Gene Bmi1
Yanyang Tu, Xingchun Gao, Gang Li, Hualin Fu, Daixiang Cui, Hui Liu, Weilin Jin, and Yongsheng Zhang

Precis: A tumor-suppressive microRNA acts by regulating a central transcriptional corepressor molecule implicated in glioblastoma, from which insights into its downstream targets in stem cell populations have emerged recently.

FOREST TRANSITION FACTORS CONTROL E2F1 TRANSCRIPTIONAL SPECIFICITY AND APOPTOTIC FUNCTION
Igor Shaib, Michael L. Gatza, Beiyou Liu, Steven P. Angus, Lingchong You, and Joseph R. Neevins

Precis: This investigation into apoptosis mechanisms suggests a rationale to combine HDAC and PI3K inhibitors as a broad-acting strategy to attack numerous types of human cancer.

ERG Is a Critical Regulator of Wnt/LEF1 Signaling in Prostate Cancer
Longtao Wu, Jonathan C. Zhao, Jung Kim, Hong-Jian Jin, Cun-Yu Wang, and Jindan Yu

Precis: This study provides a mechanistic rationale to use Wnt pathway inhibitors to treat prostate cancers that harbor a characteristic TMPRSS2-ERG genetic fusion.

Obesity Promotes Breast Cancer by CCL2-Mediated Macrophage Recruitment and Angiogenesis
Lisa M. Arendt, Jessica McCready, Patricia J. Keller, Dana D. Baker, Stephen P. Naber, Victoria Seewaldt, and Charlotte Kuperwasser

Precis: These findings developed in a novel humanized breast cancer model reveal a mechanistic role for adipocytes and macrophages that may act at early times to promote breast cancer development in obese individuals, with implications for both prevention and treatment.

CORRECTIONS

Correction: A Novel Class of Anticancer Compounds Targets the Actin Cytoskeleton in Tumor Cells

Correction: Constitutive HER2 Signaling Promotes Breast Cancer Metastasis through Cellular Senescence

Correction: PTK6 Activation at the Membrane Regulates Epithelial–Mesenchymal Transition in Prostate Cancer

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ABOUT THE COVER

Tumor cells evolve by interacting with the local microenvironment. In this study, an FGF receptor (FGFR4) is found to be pivotal for the process by which the tumor stromal microenvironment triggers conversion of epithelial cancer cells to mesenchymal phenotypes that are more invasive and metastatic. Tumor-associated fibroblasts-mediated FGFR4 activation is strongly related to a high risk of tumor metastasis and poor patient outcome, suggesting novel therapeutic opportunities for the treatment of colorectal cancer. For details, see article by Liu and colleagues on page 5926.