PD-1/SHP-2 Inhibits Tc1/Th1 Phenotypic Responses and the Activation of T Cells in the Tumor Microenvironment
Jing Li, Hyun-Bae Jie, Yu Lei, Neil Gildener-Leapman, Sumita Trivedi, Tony Green, Lawrence P. Kane, and Robert L. Ferris

Précis: This study addresses the mechanisms through which PD-1 antibody therapy restores the ability of the immune system to recognize and attack tumors by skewing intratumoral T cells toward a more potent activation phenotype that is associated with enhanced signaling and a reversal of their immunosuppressed phenotype.

Host miR155 Promotes Tumor Growth through a Myeloid-Derived Suppressor Cell–Dependent Mechanism
Suqi Chen, Long Wang, Jie Fan, Cong Ye, Donye Dominguez, Yi Zhang, Tyler J. Curiel, Deyu Fang, Timothy M. Kuzel, and Bin Zhang

Précis: These findings reveal an immunosuppressive function for an miRNA thought to be solely immunostimulatory, establishing a contextual role that prompts caution for suggested strategies to manipulate its expression for cancer therapy.

Lyosphosphatidic Acid Receptor LPAR6 Supports the Tumorigenicity of Hepatocellular Carcinoma
Antonio Mazzocca, Francesco D’Ituri, Flavia De Santis, Addolorata Filannino, Chiara Lopane, Regina C. Betz, Ying-Yi Li, Naofumi Mukaida, Peter Winter, Cosimo Tortorella, Gianluigi Gianelli, and Carlo Sabbà

Précis: These findings link overexpression of a lyosphosphatidic acid receptor during liver cancer development to upregulation of the PIM3 oncogene, which is implicated in histone modification and apoptosis, with implications for prognosis and treatment in this disease setting.

Human Pancreatic Cancer Tumors Are Nutrient Poor and Tumor Cells Actively Scavenge Extracellular Protein
Jurre J. Kamphorst, Michel Nofal, Cosimo Commisso, Sean R. Hackett, Wenyun Lu, Elda Grabocka, Matthew G. Vander Heiden, George Miller, Jeffrey A. Drebin, Dafna Bar-Sagi, Craig B. Thompson, and Joshua D. Rabinowitz

Précis: Scavenging of extracellular protein represents a previously unappreciated pathway of nutrient uptake in human pancreatic tumors, providing new insights into how these tumors grow in nutrient-poor conditions.
Gain of Glucose-Independent Growth upon Metastasis of Breast Cancer Cells to the Brain

Jinyu Chen, Ho-Jeong Lee, Xuefeng Wu, Lei Huo, Sun-Jin Kim, Lei Xu, Yan Wang, Junqing He, Lakshmi R. Bollu, Guang Cao, Fei Su, James Briggs, Xiaoqing Liu, Tamar Melman, John M. Asara, Isaiah J. Fidler, Lewis C. Cantley, Jason W. Locasale, and Zhang Weihua

Précis: This study defines a specific metabolic condition required to sustain brain metastasis, with therapeutic implications for how this deadly feature of advanced breast cancer might be eradicated.

Identification of a Functional SNP in the 3’UTR of CXCR2 That Is Associated with Reduced Risk of Lung Cancer

Bríd M. Ryan, Ana I. Robles, Andrew C. McClary, Majda Haznadar, Elise D. Bowman, Sharon R. Pine, Derek Bown, Mohammed Khan, Kouya Shiraiishi, Takashi Kohno, Hirokazu Okayama, Ramakrishna Modali, Jun Yokota, and Curtis C. Harris

Précis: A genetic polymorphism in a chemokine receptor known to drive myeloid recruitment in the tumor microenvironment confers strong protection against lung cancer, with potential implications in understanding etiology, prognosis, and therapeutic response in this widespread disease.

Perinatal and Familial Risk Factors for Brain Tumors in Childhood through Young Adulthood

Casey Crump, Jan Sundquist, Weiva Sieh, Marilyn A. Winkleby, and Kristina Sundquist

Précis: In this large national cohort study, high birth weights associated with an increased risk of brain tumors were traced to a role for high growth rates rather than gestational age.

PDGFRα and β Play Critical Roles in Mediating Foxq1-Driven Breast Cancer Stemness and Chemoresistance

Fanyan Meng, Cecilia L. Speyer, Bin Zhang, Yongzhong Zhao, Wei Chen, David H. Gorski, Fred R. Miller, and Guojun Wu

Précis: These findings establish a novel functional connection in breast cancer, with implications for ways to combine targeted therapeutics to stratify patients and enhance efficacy.

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Altered metabolism is a common feature of cancer cells, but little is known as to what metabolic changes benefit breast cancer brain metastases. Fructose-1,6-bisphosphatase 2 (FBP2), a rate limiting enzyme of gluconeogenesis, was found to be significantly upregulated in breast cancer brain metastases. FBP2 empowers brain metastatic cancer cells with the ability to grow independently of glucose. For details, see article by Chen and colleagues on page 554.

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