Tyrosine Phosphorylation of p27Kip1 Correlates with Palbociclib Responsiveness in Breast Cancer Tumor Cells Grown in Explant Culture
Susan R.S. Gottesman, Jonathan Somma, Vladislav Tsiperson, Lisa Dresner, Usha Govindarajulu, Priyank Patel, and Stacy W. Blain

Discovery and Characterization of Recurrent, Targetable ALK Fusions in Leiomyosarcoma
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Epigenetic Regulation of Dlg1, via Kaiso, Alters Mitotic Spindle Polarity and Promotes Intestinal Tumorigenesis
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Growth Factor–Independent 1 Is a Tumor Suppressor Gene in Colorectal Cancer
Min-Shan Chen, Yuan-Hung Lo, Xi Chen, Christopher S. Williams, Jessica M. Donnelly, Zachary K. Criots, Shreena Patel, Joann M. Butkus, Julien Durbelle, Milton J. Finegold, and Noah F. Shroyer

The Tumor Suppressor FBW7 and the Vitamin D Receptor Are Mutual Cofactors in Protein Turnover and Transcriptional Regulation
Reyhaneh Salehi-Tabar, Babak Memari, Hilary Wong, Vassil Dimitrov, Natacha Rochel, and John H. White

CREBBP/EP300 Bromodomain Inhibition Affects the Proliferation of AR-Positive Breast Cancer Cell Lines
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Multiclonality and Marked Branched Evolution of Low-Grade Endometrioid Endometrial Carcinoma
Lorena Lazo de la Vega, Mia C. Samaha, Kevin Hu, Nolan R. Biek, Javed Siddiqui, Daniel H. Hovelson, Chia-Jen Liu, Cody S. Carter, Kathleen R. Cho, Andrew P. Scialis, and Scott A. Tomlins

Ovarian Tumor Cell Expression of Claudin-4 Reduces Apoptotic Response to Paclitaxel
Christopher Breed, Douglas A. Hicks, Patricia G. Webb, Carly E. Galimani, Benjamin G. Bixle, Kian Behbahani, and Heidi K. Baungartner

Combinations of Tyrosine Kinase Inhibitor and ERAD Inhibitor Promote Oxidative Stress–Induced Apoptosis through ATF4 and KLF9 in Medullary Thyroid Cancer
Rozita Bagheri-Yarmand, Krishna M. Sinha, Ling Li, Yue Lu, Gilbert J. Cote, Steven I. Sherman, and Robert F. Gagel

NF-κB and Poly (ADP-ribose) Polymerase 1 Form a Positive Feedback Loop that Regulates DNA Repair in Acute Myeloid Leukemia Cells
Ding Li, Yufei Luo, Xianling Chen, LingYu Zhang, Tingting Wang, Yingting/Zhuang, Yinguai Fan, Jianhua Xu, Yuanzhong Chen, and Lixian Wu

The Antitumor Drugs Trabectedin and Lurbinectedin Induce Transcription-Dependent Replication Stress and Genome Instability
Emanuela Tumini, Emilia Herrera-Moyano, Marta San Martin-Alonso, Sonia Barroso, Carlos M. Galmarini, and Andrés Aguilera
CCL2 Is a Vascular Permeability Factor Inducing CCR2-Dependent Endothelial Retraction during Lung Metastasis
Marko Roblek, Darya Protsyuk, Paul F. Becker, Cristina Stefanescu, Christian Gorzelanny, Jesus F. Glaus Garzon, Lucia Knopfova, Mathias Heikenwalder, Bruno Luckow, Stefan W. Schneider, and Lubor Borsig

Reactive Oxygen Species (ROS)-Inducing Triterpenoid Inhibits Rhabdomyosarcoma Cell and Tumor Growth through Targeting Sp Transcription Factors
Ravi Kasiappan, Indira Jutooru, Kumaravel Mohankumar, Keshav Karki, Alexandra Lacey, and Stephen Safe

Interactions with Muscle Cells Boost Fusion, Stemness, and Drug Resistance of Prostate Cancer Cells
Berna Uygur, Evgenia Leikina, Kamran Melikov, Rafael Villasmil, Santosh K. Verma, Calvin P.H. Vary, and Leonid V. Chernomordik

Periprostatic Adipose Tissue Favors Prostate Cancer Cell Invasion in an Obesity-Dependent Manner: Role of Oxidative Stress
Victor Laurent, Aurélie Toulet, Camille Attané, Delphine Milhas, Stéphanie Dauvillier, Falek Zaidi, Emily Clement, Mathieu Cinato, Sophie Le Gonidec, Adrien Guérard, Camille Lehuédé, David Garandeau, Laurence Nieto, Edith Renaud-Gabardos, Anne-Catherine Piats, Philippe Valet, Bernard Malavaud, and Catherine Muller

Epigenetic control of cellular polarity has recently come under scrutiny as a potential driver of tumorigenesis and progression. The cover shows a whole mount immunofluorescence image of an intestinal cell organoid in which the scaffold protein Dlg1 had been disrupted (green: phalloidin; red: lysozyme; blue: DAPI). Loss of Dlg1 does not affect the cells’ ability to maintain polarity, but rather causes improper orientation of the mitotic spindle and loss of planar cell division, causing increased dwelling time in intestinal crypts. The authors suggest that delayed exit from the crypts allows for additional time to accumulate and retain mutations without increasing the overall mutation rate, thus contributing to a “tumor-permissive” environment in the intestine. Please see the article by Young and colleagues (beginning on page 686) for more information.