Breaking Advances

Highlights from Recent Cancer Literature

Reviews

Resistance to BRAF Inhibitors: Unraveling Mechanisms and Future Treatment Options
Jessie Villanueva, Adina Vultur, and Meenhard Herlyn

Diversity of Human Leukemia Xenograft Mouse Models: Implications for Disease Biology
Lüder Hinrich Meyer and Klaus-Michael Debatin

Microenvironment and Immunology

The LMP7-K Allele of the Immunoproteasome Exhibits Reduced Transcript Stability and Predicts High Risk of Colon Cancer
Barbara Fellerhoff, Songhai Gu, Barbara Laumbacher, Andreas G. Nerlich, Elisabeth H. Weiss, Jürgen Glas, Reinhard Kopp, Judith P. Johnson, and Rudolf Wank

Blockade of TGF-β Signaling by the TGFβR-1 Kinase Inhibitor LY2109761 Enhances Radiation Response and Prolongs Survival in Glioblastoma
Mengxian Zhang, Susanne Kleber, Manuel Röhrich, Carmen Timke, Na Han, Jochen Trauttenberg, Ana Martin-Villalba, Juergen Debus, Peter Peschke, Ute Wirker, Michael Lahm, and Peter E. Huber

Contents

Cancer Research
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Molecular and Cellular Pathobiology

Multiple Stress Signals Activate Mutant p53 In Vivo
Young-Ah Suh, Sean M. Post, Ana C. Elizondo-Fraire, Daniela R. Maccio, James G. Jackson, Adel K. El-Naggar, Carolyn Van Pelt, Tamara Terzian, and Guillermira Lozano

Précis: Mutant p53, like wild-type p53, is regulated by a host of cellular stimuli in vivo, and this stabilization provides the tumor with a growth advantage, manifesting in a more aggressive phenotype and decreased survival.

Egfl7 Promotes Tumor Escape from Immunity by Repressing Endothelial Cell Activation
Suzanne Delfortrie, Sébastien Pinte, Virginie Mattot, Chantal Samson, Gaëlle Villain, Bertrand Caetano, Géraldine Lauridant-Philippin, Marie-Christine Baranzelli, Jacques Bonnetterre, François Trottein, Christelle Faveeuw, and Fabrice Soncin

Précis: A secreted endothelial cell protein promotes immune escape by downregulating leukocyte adhesion molecules that mediate immune cell extravasation from blood vessels into tumors.

Classification of Epstein–Barr Virus–Positive Gastric Cancers by Definition of DNA Methylation Epigenotypes
Keisuke Matsusaka, Atsushi Kaneda, Genta Nagae, Tetsuo Ushiku, Yasuko Kikuchi, Rumi Hino, Hiroshi Uozaki, Yasuyuki Seto, Kenzo Takada, Hiroyuki Aburatani, and Masashi Fukayama

Précis: A high DNA methylation epigenotype induced by Epstein–Barr virus may play a causative role in the setting of gastric cancers.
Chronic Activation of Wild-Type Epidermal Growth Factor Receptor and Loss of Cdkn2a Cause Mouse Glioblastoma Formation
Jaime Acquaviva, Hyun Jung Jun, Julie Lessard, Rolando Ruiz, Haihao Zhu, Melissa Donovan, Steve Woolfenden, Abraham Boskovitz, Ami Raval, Roderick T. Bronson, Rolf Pfannl, Charles A. Whittaker, David E. Housman, and Al Charest

Precis: Findings show that EGFR requires chronic ligand activation for gliomagenesis to occur, and in the context of Cdkn2a loss the tumors are addicted to EGFR signaling.

Précis: Whereas epigenetic regulators constitute some appealing targets for therapeutic inhibition, this study reveals a target for which cancer stem cell selectivity may be achievable.

Identification of Tumorigenic Cells in KrasG12D-Induced Lung Adenocarcinoma
Huan-Chieh Cho, Chao-Yang Lai, Li-En Shao, and John Yu

Précis: Mitotic kinases currently thought of as oncogenes may play a supportive role in Taxol chemotherapy, suggesting that therapeutic inhibition of these kinases, which has been suggested, might actually be counterproductive for cancer treatment.

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Novel Histone Demethylase LSD1 Inhibitors Selectively Target Cancer Cells with Pluripotent Stem Cell Properties
Jing Wang, Fei Lu, Qi Ren, Hong Sun, Zhengshuang Xu, Rongfeng Lan, Yuqing Liu, David Ward, Junmin Quan, Tao Ye, and Hui Zhang

Précis: Findings provide a striking preclinical illustration of the efficiency of a targeted protein-based therapy to eradicate metastases in the lung, where many advanced human cancers spread.

Précis: Cooperative phosphorylation of FADD by Aur-A and Plk1 in response to Taxol triggers both apoptotic and necrotic cell death.

Cooperative Phosphorylation of FADD by Aur-A and Plk1 in Response to Taxol Triggers Both Apoptotic and Necrotic Cell Death
Moon-Sun Jang, Su-Jin Lee, Nam Sook Kang, and Eunhee Kim

Précis: Cooperative phosphorylation of FADD by Aur-A and Plk1 in response to Taxol triggers both apoptotic and necrotic cell death.

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Précis: Bronchiolar Clara cells are identified to be the cell of origin in KrasG12D-driven lung adenocarcinomas, a finding with implications for the detection and development of therapeutics against this group of lung cancers.

Lactoferrin–Endothelin-1 Axis Contributes to the Development and Invasiveness of Triple-Negative Breast Cancer Phenotypes
Ngoc-Han Ha, Vasudha S. Nair, Divijendra Natha Sirigiri Reddy, Prakriti Mudvari, Kazuhiro Ohshiro, Krishna Sumanth Ghanta, Suresh B. Pakala, Da-Qiang Li, Luis Costa, Allan Lipton, Rajendra A. Badwe, Suzanne Fuqua, Margareth Wallon, George C. Prendergast, and Rakesh Kumar

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Mucin Glycosylating Enzyme GALNT2 Regulates the Malignant Character of Hepatocellular Carcinoma by Modifying the EGF Receptor
Yao-Ming Wu, Chiung-Hui Liu, Rey-Heng Hu, Miao-Juei Huang, Jian-Jr Lee, Chi-Hau Chen, John Huang, Hong-Shiie Lai, Po-Huang Lee, Wen-Ming Hsu, Hsiu-Chin Huang, and Min-Chuan Huang

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Précis: This study explored the role of Wnt signaling in glioblastoma and revealed that both canonical and noncanonical pathways are required for proliferation, offering new therapeutic targets for these brain tumors.

Précis: Findings suggest that induction of glucose import is an important prosurvival function of the NF-κB pathway.

Précis: This study provides the initial preclinical validation of an adhesion-linked G protein-coupled receptor as a drug development target for prostate cancer therapy.

Correction: ΔNp63 Versatilley Regulates a Broad NF-κB Gene Program and Promotes Squamous Epithelial Proliferation, Migration, and Inflammation

ABOUT THE COVER

Mice carrying a germline p53 missense mutation equivalent to one commonly found in human cancers do not stabilize mutant p53 in healthy tissues, suggesting similarities to wild type p53 regulation. In this study, many of the same signals that contribute to the stabilization of wild type p53 (IR, ROS, p16INK4a loss, and activation of the Myc and K-Ras oncogenes) do in fact also stabilize mutant p53, often resulting in worse outcomes. This tumor sample has stable expression of mutant p53. For details, see the article by Suh and colleagues on page 7168 of this issue.
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