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IMAGING, DIAGNOSIS, PROGNOSIS

ON 01910.Na Is Selectively Cytotoxic for Chronic Lymphocytic Leukemia Cells through a Dual Mechanism of Action Involving PI3K/AKT Inhibition and Induction of Oxidative Stress
Colby M. Chapman, Xiaemeng Sun, Mark Roschewski, Georg Aue, Mohamed Farooqui, Lawrence Stennett, Federica Gibellini, Diane Arthur, Patricia Pérez-Galán, and Adrian Wiestner

CANCER THERAPY: PRECLINICAL

Impaired Cognitive Function and Hippocampal Neurogenesis following Cancer Chemotherapy
Lori-Ann Christie, Munjal M. Acharya, Vipan K. Parhiark, Anna Nguyen, Vahan Martirosian, and Charles L. Limoli

Cyclin-Dependent Kinase 7/9 Inhibitor SNS-032 Abrogates FIP1-like-1 Platelet-Derived Growth Factor Receptor α and Bcr-Abl Oncogene Addiction in Malignant Hematologic Cells
Yongbin Wu, Chun Chen, Xiaoyong Sun, Xianping Shi, Bei Jin, Ke Ding, Sai-Ching Jim Yeung, and Jingxuan Pan

18F-FDG-PET/CT Imaging as an Early Survival Predictor in Patients with Primary High-Grade Soft Tissue Sarcomas Undergoing Neoadjuvant Therapy
A Panel of Four miRNAs Accurately Differentiates Malignant from Benign Indeterminate Thyroid Lesions on Fine Needle Aspiration

Xavier M. Keutgen, Filippo Filicori, Michael J. Crowley, Yongchun Wang, Theresa Scognamiglio, Rana Hoda, Daniel Buitrago, David Cooper, Martha A. Zeiger, Rasa Zarnegar, Olivier Elemento, and Thomas J. Fahey III

Phase I/II Trial of Carboplatin and Paclitaxel Chemotherapy in Combination with Intravenous Oncolytic Reovirus in Patients with Advanced Malignancies

Eleni M. Karapanagiotou, Victoria Roulstone, Katie Twigger, Merced Ball, MaryAnne Tanay, Chris Nutting, Kate Newbold, Martin E. Gore, James Larkin, Konstantinos N. Syrigos, Matt Coffey, Brad Thompson, Karl Mettiger, Richard G. Vile, Hardev S. Pandha, Geoff D. Hall, Alan A. Melcher, John Chester, and Kevin J. Harrington

CANCER THERAPY: CLINICAL

CTLA-4 Blockade with Ipilimumab: Long-term Follow-up of 177 Patients with Metastatic Melanoma

Peter A. Prieto, James C. Yang, Richard M. Sherry, Marybeth S. Hughes, Udai S. Kammula, Donald E. White, Catherine L. Levy, Steven A. Rosenberg, and Giao Q. Phan

Phase II, Open-Label Study of Brivanib as Second-Line Therapy in Patients with Advanced Hepatocellular Carcinoma

Richard S. Finn, Yoon-Koo Kang, Mary Mulcahy, Blase N. Politte, Ho Yeong Lim, Ian Walters, Christine Baudelet, Demetrios Manekas, and Joong-Won Park

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Phase I Study of Rigosertib, an Inhibitor of the Phosphatidylinositol 3-Kinase and Polo-like Kinase 1 Pathways, Combined with Gemcitabine in Patients with Solid Tumors and Pancreatic Cancer

Wen Wee Ma, Wells A. Messersmith, Grace K. Dy, Colin D. Weekes, Amy Whitworth, Cher Ren, Manoj Maniar, Francois Wilhelm, S. Gail Eckhardt, Alex A. Adjei, and Antonio Jimeno

Sorafenib Is an Inhibitor of UGT1A1 but Is Metabolized by UGT1A9: Implications of Genetic Variants on Pharmacokinetics and Hyperbilirubinemia


Phase II Efficacy and Pharmacogenomic Study of Selumetinib (AZD6244; ARRY-142886) in Iodine-131 Refractory Papillary Thyroid Carcinoma with or without Follicular Elements


See commentary p. 1827

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PREDICTIVE BIOMARKERS AND PERSONALIZED MEDICINE

Tumor Hypoxia Predicts Biochemical Failure following Radiotherapy for Clinically Localized Prostate Cancer

Michael Milosevic, Padraig Warde, Cynthia Ménard, Peter Chung, Ants Toi, Adrian Ishkanian, Michael McLean, Melanie Pintilie, Jenna Sykes, Mary Gospodarowicz, Charles Catton, Richard P. Hill, and Robert Bristow

Implications of Plasma Protein Binding for Pharmacokinetics and Pharmacodynamics of the γ-Secretase Inhibitor RO4929097

Jiannmei Wu, Patricia M. Lofrusso, Larry H. Matherly, and Jing Li

Correction: Molecular Imaging of TGFβ-Induced Smad2/3 Phosphorylation Reveals a Role for Receptor Tyrosine Kinases in Modulating TGFβ Signaling

CORRECTIONS

Correction: Molecular Imaging of TGFβ-Induced Smad2/3 Phosphorylation Reveals a Role for Receptor Tyrosine Kinases in Modulating TGFβ Signaling
ABOUT THE COVER

High-level EGFR gene amplification can be retained in glioblastoma stem-like cell lines established and propagated without recombinant EGF. In contrast, high-level amplification is lost in parallel cell lines from the same tumors established with EGF supplementation. Cell lines with high-level EGFR amplification produce highly aggressive xenograft tumors in the brains of nude mice, retaining the EGFR amplification as shown in the cover figure, whereas counterpart cell lines, lacking high-level amplification, are either nontumorigenic or grow significantly more slowly in vivo. For details, see the article by Schulte and colleagues on page 1901 of this issue.