Highlights of This Issue 1819

SPECIAL FEATURES

CCR Translations

1821 The Antitumor Immunity of Ipilimumab: (T-cell) Memories to Last a Lifetime?
Michael A. Postow, Margaret K. Callahan, and Jedd D. Wolchok
See article p. 2039

1824 Second-Line Therapies in Hepatocellular Carcinoma: Emergence of Resistance to Sorafenib
Augusto Villanueva and Josep M. Llovet
See article p. 2090

1827 In Search of a Real "Targeted" Therapy for Thyroid Cancer
Marcia S. Brose
See article p. 2056

CCR New Strategies

1830 New Strategies for Advanced Neuroendocrine Tumors in the Era of Targeted Therapy
Mei Dong, Alexandria T. Phan, and James C. Yao

1837 Statistical Issues and Recommendations for Noninferiority Trials in Oncology: A Systematic Review
Shiro Tanaka, Yousuke Kinjo, Yoshiki Kataoka, Kenichi Yoshimura, and Satoshi Teramukai

Statistics in Clinical Cancer Research

1848 Abiraterone in Prostate Cancer: A New Angle to an Old Problem
Mark N. Stein, Susan Goodin, and Robert S. DiPaola

Molecular Pathways

1855 Molecular Pathways: Fibroblast Growth Factor Signaling: A New Therapeutic Opportunity in Cancer
A. Nigel Brooks, Elaine Kilgour, and Paul D. Smith

HUMAN CANCER BIOLOGY

1863 Molecular Pathways: Regulation and Therapeutic Implications of Multidrug Resistance
Kevin G. Chen and Branimir I. Sikic

1870 Immune Thrombocytopenia in Patients with Chronic Lymphocytic Leukemia Is Associated with Stereotyped B-cell Receptors
Carlo Visco, Francesco Maura, Giacomo Tuana, Luca Agnelli, Marta Lionetti, Sonia Fabris, Elisabetta Novella, Ilaria Giaretta, Gianluigi Reda, Wilma Barcellini, Luca Baldini, Antonio Neri, Francesco Rodeghiero, and Agostino Cortelezzi

1879 MAPKAP Kinase 2 Overexpression Influences Prognosis in Gastrointestinal Stromal Tumors and Associates with Copy Number Variations on Chromosome 1 and Expression of p58 MAP Kinase and ET1
Peter Birner, Andrea Beer, Ursula Vinatzer, Susanne Stary, Romana Höffberger, Nadine Nirtl, Fritz Wrb, Berthold Streubel, and Sebastian F. Schoppmann

1888 Protein Kinase CK2 Protects Multiple Myeloma Cells from ER Stress–Induced Apoptosis and from the Cytotoxic Effect of HSP90 Inhibition through Regulation of the Unfolded Protein Response
Sabrina Manni, Alessandra Brancalion, Laura Quotti Tubi, Anna Colpo, Laura Pavan, Anna Cabrelle, Elisa Ave, Fortunato Zaffino, Giovanni Di Maira, Maria Ruzzene, Fausto Adami, Renato Zambello, Maria Rita Pitari, Pierfrancesco Tassone, Lorenzo A. Pinna, Carmela Gurrieri, Gianpiero Semenzato, and Francesco Piazza

1901 Glioblastoma Stem–like Cell Lines with Either Maintenance or Loss of High-Level EGFR Amplification, Generated via Modulation of Ligand Concentration
Alexander Schulte, Hauke S. Gündner, Tobias Martens, Svenja Zapf, Sabine Riethdorf, Clemens Wulfing, Malgorzata Stoupiec, Manfred Westphal, and Katrin Lamszus
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 Zarmegar, 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 Mettigner, 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. Kamnula, Donald E. White, Catherine I. Levy, Steven A. Rosenberg, and Giao Q. Phan

See commentary p. 1821

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, Chen Ren, Manoj Maniar, Francois Wilhelm, S. Gail Eckhardt, Alex A. Adjei, and Antonio Jimeno

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

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, Melania Pintilie, Jenna Sykes, Mary Gospodarowicz, Charles Catton, Richard P. Hill, and Robert Bristow

PREDICTIVE BIOMARKERS AND PERSONALIZED MEDICINE

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

Jianmei Wu, Patricia M. LoRusso, Larry H. Matherly, and Jing Li

Correction: Molecular Imaging of TGFβ1-Induced Smad2/3 Phosphorylation Reveals a Role for Receptor Tyrosine Kinases in Modulating TGFβ Signaling

CORRECTIONS

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


See commentary p. 1824
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.
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18 (7)


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