Highlights of This Issue

SPECIAL FEATURES

CCR Translations

3719  Mind the Gap: Potential for Rebounds during Antiangiogenic Treatment Breaks
John M.L. Ebos and Roberto Pili
See article p. 3961

CCR Perspectives in Drug Approval

3722  Vandetanib for the Treatment of Symptomatic or Progressive Medullary Thyroid Cancer in Patients with Unresectable Locally Advanced or Metastatic Disease: U.S. Food and Drug Administration Drug Approval Summary

Statistics in Clinical Cancer Research

3731  Cure Models as a Useful Statistical Tool for Analyzing Survival
Megan Othus, Bart Barlogie, Michael L. LeBlanc, and John J. Crowley

CCR Drug Updates

3737  Crizotinib for ALK-Rearranged Non–Small Cell Lung Cancer: A New Targeted Therapy for a New Target
Leena Gandhi and Pasi A. Jänne

Molecular Pathways

3743  Molecular Pathways: Targeting P21-Activated Kinase 1 Signaling in Cancer—Opportunities, Challenges, and Limitations
Jeyanthi Eswaran, Da-Qiang Li, Anil Shah, and Rakesh Kumar

Review

3750  Combining Antiangiogenics to Overcome Resistance: Rationale and Clinical Experience
Victor Moreno Garcia, Bristi Basu, L. Rhoda Molife, and Stan B. Kaye

HUMAN CANCER BIOLOGY

3762  Tumor-Associated Macrophages in Pediatric Classical Hodgkin Lymphoma: Association with Epstein-Barr Virus, Lymphocyte Subsets, and Prognostic Impact
Mário Henrique M. Barros, Rocio Hassan, and Gerald Niedobitek

3772  Defective Epidermal Innate Immunity and Resultant Superficial Dermatophytosis in Adult T-cell Leukemia/Lymphoma
Yu Sawada, Motonobu Nakamura, Rieko Kabashima-Kubo, Takatoshi Shimauchi, Miwa Kobayashi, and Yoshihi Tokura

3780  FGFR4 Blockade Exerts Distinct Antitumorigenic Effects in Human Embryonal versus Alveolar Rhabdomyosarcoma
Lisa E.S. Crose, Katherine T. Etheridge, Candy Chen, Brian Belyea, Lindsay J. Talbot, Rex C. Bentley, and Corinne M. Linardic

3791  Integrative Genomic Analysis Implicates Gain of PIK3CA at 3q26 and MYC at 8q24 in Chronic Lymphocytic Leukemia
Jennifer R. Brown, Megan Hanna, Bethany Tesar, Lillian Werner, Nathalie Pochet, John M. Asara, Yaoyu E. Wang, Paolo dal Cin, Stacey M. Fernandes, Christina Thompson, Laura MacConaill, Catherine J. Wu, Yves Van de Peer, Mick Correll, Aviv Regev, Donna Neuberg, and Arnold S. Freedman
Development and Characterization of a Novel CD19CherryLuciferase (CD19CL) Transgenic Mouse for the Preclinical Study of B-Cell Lymphomas

Luigi Scotto, Marianna Kruthof-de Julio, Luca Paoluzzi, Matko Kalac, Enrica Marchi, Jairo Baquero Buitrago, Jennifer Amengual, Michael M. Shen, and Owen A. O’Connor

Development of a Human Monoclonal Antibody for Potential Therapy of CD27-Expressing Lymphoma and Leukemia

Laura A. Vitale, Li-Zhen He, Lawrence J. Thomas, Jennifer Widger, Jeffrey Weidlick, Andrea Crocker, Thomas O’Neill, James Storey, Martin J. Glennie, Deanna M. Grote, Stephen M. Ansell, Henry Marsh, and Tibor Keler

Elevation of c-FLIP in Castrate-Resistant Prostate Cancer Antagonizes Therapeutic Response to Androgen Receptor–Targeted Therapy

Clare McCourt, Pamela Maxwell, Roberta Mazzucchelli, Rodolfo Montironi, Marina Scarpelli, Manuel Salto-Tellez, Joe M. O’Sullivan, Daniel B. Longley, and David J.J. Waugh

Development of an Fc-Enhanced Anti–B7-H3 Monoclonal Antibody with Potent Antitumor Activity


Antitumor Activity of Targeted and Cytotoxic Agents in Murine Subcutaneous Tumor Models Correlates with Clinical Response

Harvey Wong, Edna F. Choo, Bruno Allicke, Xiao Ding, Hank L. Erin McNamara, Frank-Peter Theil, Jay Tibbatts, Lori S. Friedman, Cornels E.C.A. Hop, and Stephen E. Gould

Anti-Myeloma Effects of the Novel Anthracycline Derivative INNO-206

Eric Sanchez, Mingjie Li, Cathy Wang, Cydney M. Nichols, Jennifer Li, Haining Chen, and James R. Berenson

Brachyury, a Driver of the Epithelial–Mesenchymal Transition, Is Overexpressed in Human Lung Tumors: An Opportunity for Novel Interventions against Lung Cancer

Mario Roselli, Romaine I. Fernando, Fiorella Guadagni, Antonella Spilia, Jhessica Alessandroni, Raffaele Palmirotta, Leopoldo Costarelli, Mary Litzinger, Duane Hamilton, Bruce Huang, Joanne Tucker, Kwong-Yok Tsang, Jeffrey Schlom, and Claudia Palena

Targeting Fibroblast Growth Factor Receptor Signaling Inhibits Prostate Cancer Progression

Shu Feng, Longjiang Shao, Wendong Yu, Paul Gavine, and Michael Ittmann

Resistance to TRAIL Is Mediated by DARPP-32 in Gastric Cancer

Abbes Belkhiri, Shoumin Zhu, Zheng Chen, Mohammed Soutto, and Wael El-Rifai

GDC-0941, a Novel Class I Selective PI3K Inhibitor, Enhances the Efficacy of Docaetaxel in Human Breast Cancer Models by Increasing Cell Death In Vitro and In Vivo

Jeffrey J. Wallin, Jane Guan, Wei Wei Prior, Leslie B. Lee, Leanne Berry, Lisa D. Belmont, Hartmut Koeppen, Marcia Belvin, Lori S. Friedman, and Deepak Sampath

AT13148 Is a Novel, Oral Multi-AGC Kinase Inhibitor with Potent Pharmacodynamic and Antitumor Activity


Vandetanib, an Inhibitor of VEGF Receptor-2 and EGF Receptor, Suppresses Tumor Development and Improves Prognosis of Liver Cancer in Mice

Kinya Inoue, Takuji Torimura, Toru Nakamura, Hideki Iwamoto, Hiroshi Masuda, Mitsuhiko Abe, Osamu Hashimoto, Hiromori Koga, Takato Ueno, Hirohisa Yano, and Michio Sata
<table>
<thead>
<tr>
<th>Page No.</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>3934</td>
<td>Pyrophosphorolysis-Activated Polymerization Detects Circulating Tumor DNA in Metastatic Uveal Melanoma</td>
<td>Jordan Madic, Sophie Piperno-Neumann, Vincent Servois, Aurore Rampanou, Maud Milder, Bénédicte Trouiller, David Gentien, Stéphanie Saada, Franck Assayag, Aurélie Thuleau, Fariba Nemati, Didier Decaudin, François-Clément Biedard, Laurence Desjardins, Pascale Mariani, Olivier Lantz, and Marc-Henri Stern</td>
</tr>
<tr>
<td>3942</td>
<td>A Replication Study and Genome-Wide Scan of Single-Nucleotide Polymorphisms Associated with Pancreatic Cancer Risk and Overall Survival</td>
<td>Jason A. Willis, Sara H. Olson, Irene Orlow, Semanti Mukherjee, Robert R. McWilliams, Robert C. Kurtz, and Robert J. Klein</td>
</tr>
<tr>
<td>3972</td>
<td>Predictive Value of XRCC1 Gene Polymorphisms on Platinum-Based Chemotherapy in Advanced Non-Small Cell Lung Cancer Patients: A Systematic Review and Meta-analysis</td>
<td>Junjie Wu, Jie Liu, Yuhao Zhou, Jun Ying, Houdong Zou, Shicheng Guo, Lei Wang, Naqing Zhao, Jianjun Hu, Daru Lu, Li Jin, Qiang Li, and Jiu-Cun Wang</td>
</tr>
<tr>
<td>3982</td>
<td>Genetic Polymorphisms in MicroRNA-Related Genes as Predictors of Clinical Outcomes in Colorectal Adenocarcinoma Patients</td>
<td>Moubin Lin, Jian Gu, Cathy Eng, Lee M. Ellis, Michelle A. Hildebrandt, Jie Lin, Maosheng Huang, George A. Calin, Dingzhi Wang, Raymond N. Dulkois, Ernest T. Hawk, and XiFeng Wu</td>
</tr>
<tr>
<td>3992</td>
<td>Genetic Polymorphisms in MicroRNA-Related Genes as Predictors of Clinical Outcomes in Colorectal Adenocarcinoma Patients</td>
<td>Moubin Lin, Jian Gu, Cathy Eng, Lee M. Ellis, Michelle A. Hildebrandt, Jie Lin, Maosheng Huang, George A. Calin, Dingzhi Wang, Raymond N. Dulkois, Ernest T. Hawk, and XiFeng Wu</td>
</tr>
<tr>
<td>3998</td>
<td>Correction: A Phase I Trial of Erlotinib and Concurrent Chemoradiotherapy for Stage III and IV (M0) Squamous Cell Carcinoma of the Head and Neck</td>
<td>Yu-Yun Shao, Chien-Chung Huang, Shiou-Der Lin, Chih-Hung Hsu, and Ann-Lii Cheng</td>
</tr>
</tbody>
</table>
About the Cover

The inhibition of androgen signaling is a major therapeutic strategy in prostate cancer; however, response is often transient, and patients ultimately relapse on therapy giving rise to a currently incurable condition known as castrate-resistant prostate cancer (CRPC). McCourt and colleagues show elevated expression of the androgen-regulated antiapoptotic protein c-FLIP in prostate cancer, which is further elevated in CRPC. Repression of c-FLIP induced apoptosis in non-castrate-resistant and CRPC cells and potentiated sensitivity to AR-targeted therapy, indicating that prostate cancer cells require c-FLIP to maintain viability. Consequently, targeting c-FLIP may represent a novel strategy to improve therapeutic response to the novel antiandrogen strategies under clinical development in CRPC. For details, see the article by McCourt and colleagues on page 3822 of this issue.
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18 (14)

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