## Contents

### Highlights of This Issue 1689

#### REVIEW

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1691</td>
<td>BH3 Mimetics: Status of the Field and New Developments</td>
<td>Christian Billard</td>
</tr>
</tbody>
</table>

### SMALL MOLECULE THERAPEUTICS

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1701</td>
<td>Pharmacologic Inhibition of MEK Signaling Prevents Growth of Canine Hemangiosarcoma</td>
<td>Nicholas J. Andersen, Brian J. Nickoloff, Karl J. Dykema, Elissa A. Boguslawski, Roman I. Krivochenitser, Roe E. Froman, Michelle J. Dawes, Laurence H. Baker, Dafydd G. Thomas, Debra A. Kamstock, Barbara E. Kitchell, Kyle A. Furge, and Nicholas S. Duesbery</td>
</tr>
</tbody>
</table>

### LARGE MOLECULE THERAPEUTICS

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1774</td>
<td>Targeted Cytolysins Synergistically Potentiate Cytoplasmic Delivery of Gelonin Immunotoxin</td>
<td>Christopher M. Pirie, David V. Liu, and K. Dane Wittrup</td>
</tr>
</tbody>
</table>

### CANCER THERAPEUTICS INSIGHTS

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1796</td>
<td>Cyclin G1 Expands Liver Tumor-Initiating Cells by Sox2 Induction via Akt/mTOR Signaling</td>
<td>Wen Wen, Tao Han, Cheng Chen, Lei Huang, Wen Sun, Xue Wang, Shu-Zhen Chen, Dai-Min Xiang, Liang Tang, Dan Cao, Gen-Sheng Feng, Meng-Chao Wu, Jin Ding, and Hong-Yang Wang</td>
</tr>
</tbody>
</table>

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www.aacrjournals.org
An Autocrine Loop between TGF-β1 and the Transcription Factor Brachyury Controls the Transition of Human Carcinoma Cells into a Mesenchymal Phenotype
Cecilia Larocca, Joseph R. Cohen, Romaine I. Fernando, Bruce Huang, Duane H. Hamilton, and Claudia Palena

Impact of Tumor HER2/ERBB2 Expression Level on HER2-Targeted Liposomal Doxorubicin-Mediated Drug Delivery: Multiple Low-Affinity Interactions Lead to a Threshold Effect
Bart S. Hendriks, Stephan G. Klinz, Joseph G. Reynolds, Christopher W. Espelin, Daniel F. Gaddy, and Thomas J. Wickham

Inhibition of ABCB1 Expression Overcomes Acquired Docetaxel Resistance in Prostate Cancer
Yezl Zhu, Chengfei Liu, Nagalakshmi Nadiminty, Wei Lou, Ramakumar Tummala, Christopher P. Evans, and Allen C. Gao

Ethacrynic Acid Oxadiazole Analogs Induce Apoptosis in Malignant Hematologic Cells through Downregulation of Mcl-1 and c-FLIP, Which Was Attenuated by GSTP1-1
Guyue Liu, Rui Wang, Yuetong Wang, Pengzhan Li, Guisen Zhao, Linxiang Zhao, and Yongkui Jing

Sulindac Selectively Inhibits Colon Tumor Cell Growth by Activating the cGMP/PKG Pathway to Suppress Wnt/β-Catenin Signaling

Chk1/2 Inhibition Overcomes the Cisplatin Resistance of Head and Neck Cancer Cells Secondary to the Loss of Functional p53
Mayur A. Gadhiurkar, Maria Rita Sciuto, Marcus Vinicius Ortega Alves, Curtis R. Pickering, Abdulllah A. Osman, David M. Neskey, Mei Zhao, Alison L. Fitzgerald, Jeffrey N. Myers, and Mitchell J. Frederick

BCL-2 Hypermethylation Is a Potential Biomarker of Sensitivity to Antimitotic Chemotherapy in Endocrine-Resistant Breast Cancer

Apoptotic Circulating Tumor Cells in Early and Metastatic Breast Cancer Patients
Galatea Kallergi, Georgios Konstantinidis, Harris Markmanolaki, Maria A. Papadaki, Dimitris Mavroudis, Christos Stournaras, Vassilis Georgoulia, and Sofia Angelaki

A c-Myc Activation Sensor-Based High-Throughput Drug Screening Identifies an Antineoplastic Effect of Nitazoxanide
Hua Fan-Minogue, Sandhya Bodapati, David Solow-Cordero, Alice Fan, Ramasamy Paulmurugan, Tarik F. Massoud, Dean W. Felsher, and Sanjiv S. Gambhir

NF1 Deletion Generates Multiple Subtypes of Soft-Tissue Sarcoma That Respond to MEK Inhibition
Rebecca D. Dodd, Jeffrey K. Mito, William C. Eward, Rhea Chitalia, Mohit Sachdeva, Yan Ma, Jordi Barretina, Leslie Dodd, and David G. Kirsch

O. Michael Colvin, MD: In Memoriam (1936–2013)

Correction: MPT0B098, a Novel Microtubule Inhibitor That Destabilizes the Hypoxia-Inducible Factor-1α mRNA through Decreasing Nuclear–Cytoplasmic Translocation of RNA-Binding Protein HuR
Yun-Ching Cheng, Jing-Ping Liou, Ching-Chuan Kuo, Wen-Yang Lai, Kuang-Hsing Shih, Chi-Yen Chang, Wen-Yu Pan, Joseph T. Tseng, and Jang-Yang Chang
Correction: Dual Programmed Cell Death Pathways Induced by p53 Transactivation Overcome Resistance to Oncolytic Adenovirus in Human Osteosarcoma Cells

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ABOUT THE COVER

Continued androgen receptor (AR) expression and signaling is a key driver in castration-resistant prostate cancer (CRPC). AZD3514 is an orally bioavailable drug that inhibits androgen-dependent and -independent AR signalling \textit{in vitro} and \textit{in vivo}. Using immunohistochemistry, R3327H prostate tumors were scored for intensity of nuclear AR to assess the impact of AZD3514 on AR. For more details, see article by Loddick and colleagues on page 1715.