### Highlights of This Issue 119

#### REVIEW

**Current Preclinical Models for the Advancement of Translational Bladder Cancer Research**

David J. DeGraff, Victoria L. Robinson, Jay B. Shah, William D. Brandt, Guru Sonpavde, Yibin Kang, Monica Liebert, Xue-Ru Wu, and John A. Taylor III for the Translational Science Working Group of the Bladder Advocacy Network Think Tank

**CHEMICAL THERAPEUTICS**

**The Tyrosine Kinase Inhibitor E-3810 Combined with Paclitaxel Inhibits the Growth of Advanced-Stage Triple-Negative Breast Cancer Xenografts**

Ezia Bello, Giulia Taraboletti, Gennaro Colella, Massimo Zucchetti, Daniele Forestieri, Simonetta A. Licandro, Alexander Berndt, Petra Richter, Maurizio D’Incalci, Ennio Cavalletti, Raffaella Giavazzi, Gabriella Camboni, and Giovanna Damia

**WEE1 Kinase Inhibition Enhances the Radiation Response of Diffuse Intrinsic Pontine Gliomas**

Viola Caretti, Lotte Hiddingh, Tonny Lagerweij, Pepijn Schellen, Phil W. Koken, Esther Hulleman, Dannis G. van Vuurden, W. Peter Vandertop, Gertjan J.L. Kaspers, David P. Noske, and Thomas Wurdinger

**VS-5584, a Novel and Highly Selective PI3K/mTOR Kinase Inhibitor for the Treatment of Cancer**

Stefan Hart, Veronica Novotny-Diermayr, Kee Chuan Goh, Meredith Williams, Yong Cheng Tan, Lai Chun Ong, Albert Cheong, Bee Kheng Ng, Chithra Amalini, Babita Madan, Harish Nagaraj, Ramesh Jayaraman, Khalid M. Pasha, Kantharaj Ethirajulu, Wee Joo Chng, Nurulhuda Mustafa, Boon Cher Goh, Cyril Benes, Ulan McDermott, Mathew Garnett, Brian Dymock, and Jeanette M. Wood

#### CANCER THERAPEUTIC INSIGHTS

**Pharmacologic Blockade of FAK Autophosphorylation Decreases Human Glioblastoma Tumor Growth and Synergizes with Temozolomide**

Vita M. Golubovskaya, Grace Huang, Baotran Ho, Michael Yemma, Carl D. Morrison, Jisook Lee, Brian F. Eliezeiri, and William G. Cance

**Induction of Endoplasmic Reticulum Stress by Sorafenib and Activation of NF-κB by Lestaurtinib as a Novel Resistance Mechanism in Hodgkin Lymphoma Cell Lines**

Meike Stefanie Holz, Angela Janning, Christoph Renné, Stefan Gattenloher, Tilmann Spieker, and Andreas Bräuninger

**Triptolide Inhibits MDM2 and Induces Apoptosis in Acute Lymphoblastic Leukemia Cells through a p53-Independent Pathway**

Mei Huang, Hailong Zhang, Tao Liu, Dan Tian, Lubing Gu, and Muxiang Zhou

**Bortezomib Sensitizes Human Acute Myeloid Leukemia Cells to All-Trans-Retinoic Acid-Induced Differentiation by Modifying the RARα/STAT1 Axis**

Meidan Ying, Xinglu Zhou, Like Zhong, Nengming Lin, Hui Jing, Pishua Luo, Xiaochun Yang, Hua Song, Bo Yang, and Qiaojun He

**miRNA-100 Inhibits Human Bladder Urothelial Carcinogenesis by Directly Targeting mTOR**

Chuanliang Xu, Qinsong Zeng, Weidong Xu, Li Jiao, Yangqiong Chen, Zhenheng Zhang, Chengyao Wu, Taile Jin, Anyn Pan, Rongchao Wei, Bo Yang, and Yinghao Sun

#### COMPANION DIAGNOSTICS AND BIOMARKERS

**EGFR Exon 20 Insertion Mutations in Lung Adenocarcinomas: Prevalence, Molecular Heterogeneity, and Clinico-pathologic Characteristics**

Maria E. Arcila, Khedoudja Nafa, Jamie E. Chaft, Natasha Rekhtman, Christopher Lau, Boris A. Reva, Maureen F. Zakowski, Mark G. Kris, and Marc Ladanyi
Antitumor Activity and Pharmacodynamic Biomarkers of a Novel and Orally Available Small-Molecule Antagonist of Inhibitor of Apoptosis Proteins

Hiroyuki Sumi, Masato Yabuki, Kenichi Iwai, Megumi Morimoto, Ryosuke Hibino, Masakazu Inazuka, Kentaro Hashimoto, Yohei Kosugi, Kazunobu Aoyama, Shunsuke Yamamoto, Mie Yoshimatsu, Hideki Yamasaki, Ryuichi Tozawa, Tomoyasu Ishikawa, and Sei Yoshida

Correction: TPI-287, a New Taxane Family Member, Reduces the Brain Metastatic Colonization of Breast Cancer Cells

ABOUT THE COVER

In silico molecular modeling of the EGFR exon 20 A763_Y764 insertion mutation. Mutations in this region are predicted to cause significant rearrangement of the C helix (yellow) but do not affect the erlotinib binding pocket directly (erlotinib shown in green). Insertions in EGFR exon 20 that are more distal (3') are expected to result in a greater obstructive effect on erlotinib binding. These predictions suggest a basis for the observed variability of response to EGFR inhibition in patients with different types of EGFR exon 20 insertions. For details, see article by Arcila and colleagues on page 220.