## Table of Contents

### Highlights of This Issue  295

### REVIEW

297  **Insidious Changes in Stromal Matrix Fuel Cancer Progression**  
Fayth L. Miles and Robert A. Sikes

### CELL CYCLE AND SENESCENCE

313  **miR-338-3p Suppresses Gastric Cancer Progression through a PTEN-AKT Axis by Targeting P-REX2a**  
Bo Guo, Liying Liu, Jiayi Yao, Ruili Ma, Dongmin Chang, Zongfeng Li, Tusheng Song, and Chen Huang

322  **PAI-1 Leads to G1-Phase Cell-Cycle Progression through Cyclin D3/cdk4/6 Upregulation**  
Evan Gomes Giacoia, Makito Miyake, Adrienne Lawton, Steve Goodison, and Charles J. Rosser

### CELL DEATH AND SURVIVAL

335  **Knockdown of CABYR-a/b Increases Chemosensitivity of Human Non–Small Cell Lung Cancer Cells through Inactivation of Akt**  
Zunlei Qian, Min Li, Rui Wang, Qiangqian Xiao, Jing Wang, Mingying Li, Dacheng He, and Xueyuan Xiao

348  **T-Type Ca\(^{2+}\) Channel Inhibition Induces p53-Dependent Cell Growth Arrest and Apoptosis through Activation of p38-MAPK in Colon Cancer Cells**  
Barbara Dziegielewska, David L. Brautigan, James M. Larner, and Jaroslaw Dziegielewski

### CHROMATIN, GENE, AND RNA REGULATION

359  **RNA-Binding Protein RBM24 Regulates p63 Expression via mRNA Stability**  
Enshun Xu, Jin Zhang, Min Zhang, Yuqian Jiang, Seong-Jun Cho, and Xinbin Chen

370  **DNA Damage-Binding Complex Recruits HDAC1 to Repress Bcl-2 Transcription in Human Ovarian Cancer Cells**  
Ran Zhao, Chunhua Han, Eric Eisenhauer, John Kroger, Weiqiang Zhao, Jianhua Yu, Karuppaiyah Selvendiran, Xingluo Liu, Altai A. Wani, and Qi-En Wang

### DNA DAMAGE AND REPAIR

381  **Triapine Disrupts CtIP-Mediated Homologous Recombination Repair and Sensitizes Ovarian Cancer Cells to PARP and Topoisomerase Inhibitors**  
Z. Ping Lin, Elena S. Ratner, Margaret E. Whicker, Yashang Lee, and Alan C. Sartorelli

### GENOMICS

394  **AKT-Induced Tamoxifen Resistance Is Overturned by RRM2 Inhibition**  
Khyati N. Shah, Kshama R. Mehta, David Peterson, Marie Evangelista, John C. Livesey, and Jesika S. Faridi

408  **NF-κB Activation-Induced Anti-apoptosis Renders HER2-Positive Cells Drug Resistant and Accelerates Tumor Growth**  
Shannon T. Bailey, Penelope L. Miron, Yoon J. Choi, Bose Kochupurakkal, Gautam Maulik, Scott J. Rodig, Ruiliang Tian, Kathleen M. Foley, Teresa Bowman, Alexander Miron, Myles Brown, J. Dirk. Igleshart, and Debatij K. Bisswas

421  **Hypoxia-Independent Gene Expression Mediated by SOX9 Promotes Aggressive Pancreatic Tumor Biology**  
Peter Camaj, Carsten Jäckel, Stefan Krebs, Enrico N. DeToni, Helmut Blum, Karl-Walter Jauch, Peter J. Nelson, and Christiane J. Bruns

### ONCOGENES AND TUMOR SUPPRESSORS

433  **PP6C Hotspot Mutations in Melanoma Display Sensitivity to Aurora Kinase Inhibition**  
Actin-Binding Protein, Espin: A Novel Metastatic Regulator for Melanoma
Takeshi Yanagishita, Ichiro Yajima, Mayuko Kumasaka, Yoshiyuki Kawamoto, Toyonori Tsuzuki, Yoshinari Matsumoto, Daisuke Watanabe, and Masashi Kato

SIGNAL TRANSDUCTION

BRAFV600E Cooperates with PI3K Signaling, Independent of AKT, to Regulate Melanoma Cell Proliferation
Jillian M. Silva, Christina Bulman, and Martin McMahon

The Adherens Junction Protein Afadin Is an AKT Substrate that Regulates Breast Cancer Cell Migration
Sivan Elloul, Dmitriy Kedrin, Nicholas W. Knoblauch, Andrew H. Beck, and Alex Toker

ABOUT THE COVER

The cover image shows immunofluorescence staining of normal breast tissue obtained from surgical specimens. The staining was performed with anti-Afadin (red channel), anti-E-cadherin (green channel), and DAPI (blue channel). The staining pattern reveals Afadin staining that is largely membrane restricted and excluded from nuclei. By contrast, in surgical specimens obtained from invasive breast carcinoma, Afadin shows a predominately nuclear expression pattern with near complete loss of membrane localization. For more information, see the article by Elloul and colleagues, beginning on page 464 in this issue. In this study, the authors conclude that nuclear translocation of Afadin, mediated by the PI3K and Akt pathway, is associated with enhanced breast cancer cell migration and in turn, tumor progression.