

## Highlights of This Issue 555

### CELL CYCLE AND SENESCENCE

557 | **Telomerase Suppresses Formation of ALT-Associated Single-Stranded Telomeric C-Circles**

Matthew J. Plantinga, Kara M. Pascarelli, Anna S. Merkel, Alexander J. Lazar, Margaret von Mehren, Dina Lev, and Dominique Broccoli

### CELL DEATH AND SURVIVAL

568 | **PPP2R2C Loss Promotes Castration-Resistance and Is Associated with Increased Prostate Cancer-Specific Mortality**

Eric G. Bluemn, Elysia Sophie Spencer, Brigham Mecham, Ryan R. Gordon, Ilsa Coleman, Daniel Lewinshtein, Elahe Mostaghel, Xiaotun Zhang, James Annis, Carla Grandori, Christopher Porter, and Peter S. Nelson

579 | **Metabolic Alterations in Lung Cancer-Associated Fibroblasts Correlated with Increased Glycolytic Metabolism of the Tumor**

Virendra K. Chaudhri, Gregory G. Salzler, Salihah A. Dick, Melanie S. Buckman, Raffaella Sordella, Edward D. Karoly, Robert Mohny, Brendon M. Stiles, Olivier Elemento, Nasser K. Altorki, and Timothy E. McGraw

### CHROMATIN, GENE, AND RNA REGULATION

593 | ***mda-7/IL-24* Expression Inhibits Breast Cancer through Upregulation of Growth Arrest-Specific Gene 3 (*gas3*) and Disruption of  $\beta$ 1 Integrin Function**



You-Jun Li, Guodong Liu, Yanmei Li, Laura M. Vecchiarelli-Federico, Jeff C. Liu, Eldad Zacksenhaus, Sze W. Shan, Burton B. Yang, Qi Li, Rupesh Dash, Paul B. Fisher, Michael C. Archer, and Yaacov Ben-David

604 | **Noncanonical Regulation of the Hedgehog Mediator *GLI1* by c-MYC in Burkitt Lymphoma**

Joon Won Yoon, Marisa Gallant, Marilyn LG Lamm, Stephen Iannaccone, Karl-Frederic Vieux, Maria Proytcheva, Elizabeth Hyjek, Philip Iannaccone, and David Walterhouse

### GENOMICS

616 | **RAS/MEK-Independent Gene Expression Reveals BMP2-Related Malignant Phenotypes in the *Nf1*-Deficient MPNST**

Daochun Sun, Ramsi Haddad, Janice M. Kraniak, Steven D. Horne, and Michael A. Tainsky

### ONCOGENES AND TUMOR SUPPRESSORS

628 | **CDCP1 Regulates the Function of MT1-MMP and Invadopodia-Mediated Invasion of Cancer Cells**

Yuri Miyazawa, Takamasa Uekita, Yuumi Ito, Motoharu Seiki, Hideki Yamaguchi, and Ryuichi Sakai

638 | **Human Lung Epithelial Cells Progressed to Malignancy through Specific Oncogenic Manipulations**

Mitsuo Sato, Jill E. Larsen, Woochang Lee, Han Sun, David S. Shames, Maithili P. Dalvi, Ruben D. Ramirez, Hao Tang, John Michael DiMaio, Boning Gao, Yang Xie, Ignacio I. Wistuba, Adi F. Gazdar, Jerry W. Shay, and John D. Minna

### SIGNAL TRANSDUCTION

651 | **The Tyrosine Phosphatase SHP2 Regulates Focal Adhesion Kinase to Promote EGF-Induced Lamellipodia Persistence and Cell Migration**

Zachary R. Hartman, Michael D. Schaller, and Yehenev M. Agazie

665 **Deacetylated GM3 Promotes uPAR-Associated Membrane Molecular Complex to Activate p38 MAPK in Metastatic Melanoma**

Qiu Yan, Daniel Q. Bach, Nandita Gatla, Ping Sun, Ji-Wei Liu, Jian-Yun Lu, Amy S. Paller, and Xiao-Qi Wang

676 **Systems Analysis of the NCI-60 Cancer Cell Lines by Alignment of Protein Pathway Activation Modules with "-OMIC" Data Fields and Therapeutic Response Signatures**

Giulia Federici, Xi Gao, Janusz Slawek, Tomasz Arodz, Amanuel Shitaye, Julia D. Wulfschlegel, Ruggero De Maria, Lance A. Liotta, and Emanuel F. Petricoin III

**CORRECTION**

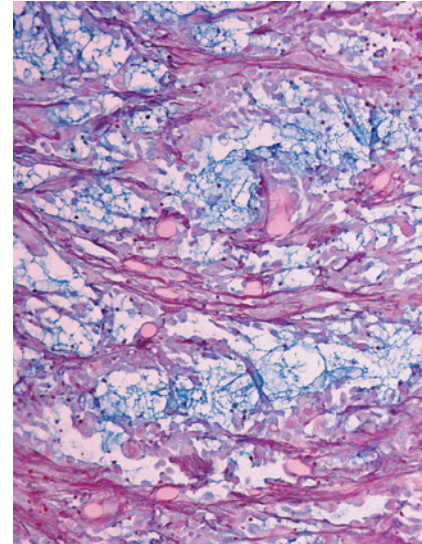
686 **Correction: Analysis of mRNA Profiles after MEK1/2 Inhibition in Human Pancreatic Cancer Cell Lines Reveals Pathways Involved in Drug Sensitivity**

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**ABOUT THE COVER**

Nonmalignant immortalized human bronchial epithelial cells (HBECs, containing hTERT and CDK4 providing a bypass of p16 – two common oncogenic changes found in lung cancer) can be progressed to fully malignant cells capable of *in vivo* tumor formation following the introduction of defined oncogenic alterations (such as high levels of oncogenic KRAS, p53 knockdown, with or without exogenous c-myc expression) mimicking genetic alterations commonly found in non-small cell lung cancer. Recent genomic data shows large number of sequence altering mutations in human lung cancers requiring sorting out of "passenger" and "driver" oncogenic changes. The HBEC preclinical *in vitro* model provides a way to test these candidates and identify the minimal, most crucial set of genetic alterations required for full malignant transformation of a bronchial epithelial cell. NOD/SCID xenograft tumors of cells with similar oncogenic changes amazingly led to multiple different lung cancer histologies (adenocarcinoma, squamous carcinoma, adeno-squamous and large cell carcinoma) indicating that factors other than oncogenic changes dictate histology. Shown here is one of the HBEC<sup>p53, KRAS</sup> tumors that differentiated into typical lung adenocarcinoma as demonstrated by Alcian-Blue Periodic Acid Schiff (PAS) stain where mucins are stained with Alcian blue (original magnification at 10X). For examples of the other histologies and details, see article by Sato, Larsen, and colleagues on page 638.



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11 (6)

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