Highlights of This Issue 565

REVIEW

The Long (lncRNA) and Short (miRNA) of It: TGFβ-Mediated Control of RNA-Binding Proteins and Noncoding RNAs
Harinarayanan Janakiraman, Reniqua P. House, Vamsi K. Gangaraju, J. Alan Diehl, Philip H. Howe, and Viswanathan Palanisamy

BET Proteins Exhibit Transcriptional and Functional Opposition in the Epithelial-to-Mesenchymal Transition
Guillaume P. Andrieu and Gerald V. Denis

Mitotically-Associated lncRNA (MANCR) Affects Genomic Stability and Cell Division in Aggressive Breast Cancer
Kirsten M. Tracy, Coralee E. Tye, Prachi N. Ghule, Heidi L.H. Malaby, Jason Stumpff, Janet L. Stein, Gary S. Stein, and Jane B. Lian

Fxo-dependent Par-4 Upregulation Prevents Long-term Survival of Residual Cells Following PI3K–Akt Inhibition
Jeffrey S. Damrauer, Stephanie N. Phelps, Katie Amuchastegui, Ryan Lupo, Nathaniel W. Mabe, Andrea Walens, Benjamin R. Kroger, and James V. Alvarez

BRD1-Mediated Acetylation Promotes Integrin αV Gene Expression Via Interaction with Sulfatide
Qian Qian Cai, Yi Wei Dong, Bing Qi, Xiaoying Shao, Rong Wang, Zhong Yi Chen, Bao Mei He, and Xing Zhong Wu

Histone H3.3K27M Mobilizes Multiple Cancer/Testis (CT) Antigens in Pediatric Glioma
Houliang Deng, Jiannming Zeng, Ting Zhang, Longcai Gong, Hongjie Zhang, Edwin Cheung, Chris Jones, and Gang Li

Overt Increase of Oxidative Stress and DNA Damage in Murine and Human Colitis and Colitis-Associated Neoplasia
Adrian Frick, Vineeta Khare, Gregor Paul, Michaela Lang, Franziska Ferk, Siegfried Knausmüller, Andrea Beer, Georg Oberhuber, and Christoph Gasche

Multigene Profiling of CTCs in mCRPC Identifies a Clinically Relevant Prognostic Signature

Microenvironment-Derived Regulation of HIF Signaling Drives Transcriptional Heterogeneity in Glioblastoma Multiforme
Dieter Henrik Heiland, Annette Gaebelein, Melanie Borries, Jakob Wörner, Nils Pompe, Pamela Franco, Sabrina Heynckes, Mark Bartholomae, Darren O. Ailín, Maria Stella Carro, Marco Prinz, Stefan Weber, Irina Mader, Daniel Delev, and Oliver Schnell

Characterization and Evidence of the miR-888 Cluster as a Novel Cancer Network in Prostate
Tsuyoshi Hasegawa, Garrison J. Glavich, Mary Pahuski, Aleena Short, O. John Semmes, Lifang Yang, Vitold Galkin, Richard Drake, and Aurora Esquela-Kerscher
The mTOR Targets 4E-BP1/2 Restrain Tumor Growth and Promote Hypoxia Tolerance in PTEN-driven Prostate Cancer
Mei Ding, Theodorus H. Van der Kwast, Ravi N. Vellanki, Warren D. Foltz, Trevor D. McKee, Nahum Sonenberg, Pier P. Pandolfi, Marianne Koritzinsky, and Bradly G. Wouters

Methylation of the HOXA10 Promoter Directs miR-196b-5p–Dependent Cell Proliferation and Invasion of Gastric Cancer Cells

Cancer Stem Cell Phenotypes in ER+ Breast Cancer Models Are Promoted by PELP1/AIB1 Complexes
Thu H. Truong, Hsiangyu Hu, Nuri A. Temiz, Kyla M. Hagen, Brian J. Girard, Nicholas J. Brady, Kathryn L. Schwertfeger, Carol A. Lange, and Julie H. Ostrander

AR Expression in Breast Cancer CTCs Associates with Bone Metastases

Methylation of the HOXA10 Promoter Directs miR-196b-5p–Dependent Cell Proliferation and Invasion of Gastric Cancer Cells

Agonist-induced CXCR4 and CB2 Heterodimerization Inhibits Ga13/RhoA-mediated Migration
Kisha A. Scarlett, El-Shaddai Z. White, Christopher J. Coke, Jada R. Carter, Latoya K. Bryant, and Cimona V. Hinton

Correction: Heparanase Promotes Glioma Progression and Is Inversely Correlated with Patient Survival

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ABOUT THE COVER
This Rapid Impact, by Andrieu and Denis (beginning on page 580), establishes that bromodomain and extraterminal (BET) proteins exert distinct and opposing transcriptional controls on EMT in breast cancer. The EMT is a developmental program that cancer cells often activate to acquire a highly plastic phenotype, notably eliciting metastasis, adaptation to the environment or chemoresistance. The cover image is an artistic rendering that shows epithelial breast cancer cells, exhibiting tight junctions enriched in the protein E-cadherin (staining). Cancer cells undergoing EMT lose their cuboidal shape, disassemble their tight junctions, hallmarks of their epithelial nature, to acquire a plastic mesenchymal phenotype. The study shows that BET proteins functionally oppose each other in the regulation of EMT. BRD2 positively controls several key EMT transcription programs, whereas BRD3 and BRD4 repress them. BET protein inhibitors obtained promising results for the treatment of some cancers, including breast cancer, yet little is known about the individual functions of each member of this particular family. To this end, more research is needed and will lead to the elaboration of improved strategies for cancer treatment.