Highlights of This Issue 687

CELL CYCLE AND SENESCENCE

Interactions between SAP155 and FUSE-Binding Protein-Interacting Repressor Bridges c-Myc and P27Kip1 Expression
Kazuyuki Matsushita, Mai Tamura, Nobuko Tanaka, Takeshi Tomonaga, Hisahiro Matsubara, Hideaki Shimada, David Levens, Liusheng He, Juhong Liu, Minoru Yoshida, and Fumio Nomura

ONCOGENES AND TUMOR SUPPRESSORS

RASSF1A-Mediated Regulation of AREG via the Hippo Pathway in Hepatocellular Carcinoma
Ei Yong Ahn, Ji Su Kim, Gi Jeong Kim, and Young Nyun Park

CELL DEATH AND SURVIVAL

Let-7c Governs the Acquisition of Chemo- or Radioresistance and Epithelial-to-Mesenchymal Transition Phenotypes in Docetaxel-Resistant Lung Adenocarcinoma
Shi-Yun Cui, Jia-Yuan Huang, Yi-Tian Chen, Hai-Zhu Song, Bing Feng, Gui-Chun Huang, Rui Wang, Long-Bang Chen, and Wei De

SIGNAL TRANSDUCTION

Activation of the FGF2-FGFR1 Autocrine Pathway: A Novel Mechanism of Acquired Resistance to Gefitinib in NSCLC
Hideki Terai, Kenzo Soejima, Hiroyuki Yasuda, Sohei Nakayama, Junko Hamamoto, Daisuke Arai, Kota Ishioka, Keiko Ohgino, Shinnosuke Ikemura, Takashi Sato, Satoshi Yoda, Ryouko Satomi, Katsuhiko Naoki, and Tomoko Betsuyaku

CHROMATIN, GENE, AND RNA REGULATION

Epigenetic Control of NF-κB-Dependent FAS Gene Transcription during Progression of Myelodysplastic Syndromes
Sandrine Ettou, Catherine Humbrecht, Blandine Benet, Katy Billot, Diane d’Allard, Virginie Mariot, Michele Goodhardt, Olivier Kosmider, Patrick Mayeur, Eric Solary, and Michaela Fontenay

GENOMICS

ERK and AKT Signaling Drive MED1 Overexpression in Prostate Cancer in Association with Elevated Proliferation and Tumorigenicity
Feng Jin, Shazia Irshad, Wei Yu, Madesh Belakavadi, Marina Chekmareva, Michael M. Ittmann, Cory Abate-Shen, and Joseph D. Fondell

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ABOUT THE COVER

In prostate epithelial cells, MED1 serves as a key transcriptional coactivator for the androgen receptor and other signal-activated transcription factors. MED1 can be phosphorylated by ERK and AKT, kinases that are commonly hyperactivated in prostate cancers as a function of tumor progression. MED1 phosphorylation significantly stabilizes its nuclear half-life and stimulates its transcriptional coactivator activity. Jin and colleagues used immunohistochemistry to characterize MED1 levels in an Nkx3.1:Ptenu-mutant mouse model of prostate cancer that recapitulates the human disease. Importantly, Nkx3.1:Ptenu-mutant prostate cancers are genetically programmed to hyperactivate ERK and AKT signaling in parallel with cancer progression. The cover shows MED1 overexpression in an Nkx3.1:Ptenu-mutant prostate adenocarcinoma that was resistant to castration. For additional results and details, please see the article by Jin and colleagues on page 736 of this issue.