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, Luisheng He, Juhong Liu, Minoru Yoshida, and Fumio Nomura

Let-7c Governs the Acquisition of Chemo- or Radioreistance 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

Oncogenic MUC1-C Promotes Tamoxifen Resistance in Human Breast Cancer
Akriti Kharbanda, Hasan Rajabi, Caining Jin, Deepak Raina, and Donald Kufe

Epigenetic Control of NF-kB-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 Mayeux, Eric Solary, and Michaela Fontenay

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

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

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, Ryozuke Satomi, Katsuhiko Naoki, and Tomoko Betsuyaku

Stem Cell Marker Nestin Is Critical for TGF-β1-Mediated Tumor Progression in Pancreatic Cancer
Huei-Ting Su, Ching-Chieh Weng, Pi-Jung Hsiao, Li-Hua Chen, Tsu-Lei Kuo, Yu-Wen Chen, Kung-Kai Kuo, and Kuang-Hung Cheng

CYP1A1 Regulates Breast Cancer Proliferation and Survival
Mariangellys Rodriguez and David A. Potter

Bioactive Lipids S1P and C1P Are Prometastatic Factors in Human Rhabdomyosarcoma, and Their Tissue Levels Increase in Response to Radio/Chemotherapy
Gabriela Schneider, Ewa Bryndza, Ahmed Abdel-Latif, Janina Ratajczak, Magdalena Maj, Maciej Tarnowski, Yuri M. Glyachkin, Peter Houghton, Andrew J. Morris, Axel Vater, Sven Klussmann, Magdalena Kucia, and Mariusz Z. Ratajczak
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:Pten-mutant mouse model of prostate cancer that recapitulates the human disease. Importantly, Nkx3.1:Pten-mutant prostate cancers are genetically programmed to hyperactivate ERK and AKT signaling in parallel with cancer progression. The cover shows MED1 overexpression in a Nkx3.1:Pten-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.
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