Highlights of This Issue 1085

REVIEW

1087  Targeting Wnts at the Source—New Mechanisms, New Biomarkers, New Drugs
Babita Madan and David M. Virshup

SMALL MOLECULE THERAPEUTICS

1095  P7170: A Novel Molecule with Unique Profile of mTORC1/C2 and Activin Receptor-like Kinase 1 Inhibition Leading to Antitumor and Antiangiogenic Activity

1107  Hsp27 Inhibition with OGX-427 Sensitizes Non–Small Cell Lung Cancer Cells to Erlotinib and Chemotherapy
Barbara Lelj-Garolla, Masafumi Kumano, Eliana Beraldi, Lucia Nappi, Palma Rocchi, Diana N. Ionescu, Ladan Fazli, Amina Zoubeidi, and Martin E. Gleave

1117  p53 Family Members Regulate Phenotypic Response to Aurora Kinase A Inhibition in Triple-Negative Breast Cancer
John J. Tentler, Anastasia A. Ionkina, Aik Choon Tan, Timothy P. Newton, Todd M. Pitts, Magdalena J. Glogowska, Peter Kabos, and Martin E. Gleave

LARGE MOLECULE THERAPEUTICS

1130  High Turnover of Tissue Factor Enables Efficient Intracellular Delivery of Antibody–Drug Conjugates

1141  Characterization of ABT-806, a Humanized Tumor-Specific Anti-EGFR Monoclonal Antibody

CANCER BIOLOGY AND SIGNAL TRANSDUCTION

1152  βIII-Tubulin Regulates Breast Cancer Metastases to the Brain
Deepak Kanojia, Ramin A. Morshed, Lingjiao Zhang, Jason M. Miska, Jian Qiao, Julius W. Kim, Peter Pytel, Irina V. Balyasnikova, Maciej S. Lesniak, and Atique U. Ahmed

1162  HOTAIR Long Noncoding RNA Promotes Gastric Cancer Metastasis through Suppression of Poly r(C)-Binding Protein (PCBP) 1
Zi-Zhen Zhang, Zhi-Yong Shen, Ya-Ying Shen, En-Hao Zhao, Jing Wang, Chao-Jie Wang, Hui Cao, and Jia Xu

1171  Radiosensitization of Primary Human Glioblastoma Stem-like Cells with Low-Dose AKT Inhibition
Monal Mehta, Atif Khan, Shabbar Danish, Bruce G. Haffly, and Hatem E. Sabaawy

1181  The Tyrosine Kinase Inhibitors Imatinib and Dasatinib Reduce Myeloid Suppressor Cells and Release Effector Lymphocyte Responses
Lisa Christiansson, Stina Söderlund, Sara Mangsbo, Henrik Hjorth-Hansen, Martin Höglund, Berit Markevam, Johan Richter, Leif Stenke, Satu Mustjoki, Angelica Loskog, and Ulla Olsson-Strömberg

1202  Endostatin Has ATPase Activity, Which Mediates Its Antiangiogenic and Antitumor Activities
Shan Wang, Xin-an Lu, Peng Liu, Yan Fu, Lin Jia, Shunli Zhan, and Yongzhang Luo

1202  Frequent Loss of NISCH Promotes Tumor Proliferation and Invasion in Ovarian Cancer via Inhibiting the FAK Signal Pathway
Jing Li, Xiaoying He, Ruofan Dong, Yuan Wang, Jinjin Yu, and Haijeng Qiu
Synthetic Lethal Screens Identify Vulnerabilities in GPCR Signaling and Cytoskeletal Organization in E-Cadherin–Deficient Cells


Loss of Tuberous Sclerosis Complex 2 (TSC2) Is Frequent in Hepatocellular Carcinoma and Predicts Response to mTORC1 Inhibitor Everolimus

Hung Huynh, Hua-Xiang Hao, Stephen L. Chan, David Chen, Richard Ong, Khee Chee Soo, Panisa Pochanard, David Yang, David Ruddy, Manway Liu, Adnan Derti, Marissa N. Balak, Michael R. Palmer, Yan Wang, Benjamin H. Lee, Dalila Sellami, Andrew X. Zhu, Robert Schlegel, and Alan Huang

MGMT Expression Predicts PARP-Mediated Resistance to Temozolomide

Oihane Erice, Michael P. Smith, Rachel White, Ibai Goicoechea, Jorge Barriuso, Chris Jones, Geoffrey P. Margison, Juan C. Acosta, Claudia Wellbrook, and Imanol Arozarena

Enhancement of the Proapoptotic Properties of Newcastle Disease Virus Promotes Tumor Remission in Syngeneic Murine Cancer Models

Sara Cuadrado-Castano, Juan Ayllon, Mena Mansour, Janis de la Iglesias-Vicente, Stefan Jordan, Shashank Tripathi, Adolfo Garcia-Sastre, and Enrique Villar

RNA Interference Using c-Myc–Conjugated Nanoparticles Suppresses Breast and Colorectal Cancer Models


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ABOUT THE COVER

The newly generated Newcastle disease virus rNDV-B1/Fas encodes the human TNF receptor Fas. Human and murine cancer cells infected by rNDV-B1/Fas displayed a modified cell death response compared with the wild-type rNDV-B1, characterized by an earlier and enhanced apoptosis response due to the overexpression of Fas following the coactivation of both extrinsic and intrinsic apoptosis pathways. The enhanced cytotoxicity shown in vitro correlated with an improved oncolytic activity and therapeutic effect of rNDV-B1/Fas virus in intratumoral-treated melanoma-bearing mice. The cover image shows murine B16-F10 melanoma cells infected by rNDV-B1/Fas virus. The human Fas receptor (red) is expressed on the surface of infected cells (green) but also internalized following induction of apoptosis. DNA was counterstained with Hoechst. For details, see the article by Cuadrado-Castano and colleagues on page 1247.