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JAK–STAT and JAK–PI3K–mTORC1 Pathways Regulate Telomerase Transcriptionally and Posttranslationally in ATL Cells
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Obatoclax Interacts Synergistically with the Irreversible Proteasome Inhibitor Carfilzomib in GC- and ABC-DLBCL Cells In Vitro and In Vivo
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The Effect of Different Linkers on Target Cell Catabolism and Pharmacokinetics/Pharmacodynamics of Trastuzumab Maytansinoid Conjugates
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ERK Inhibition Overcomes Acquired Resistance to MEK Inhibitors
Georgia Hatzivassiliou, Bonnie Liu, Carol O'Brien, Jill M. Spoerke, Klaus P. Hoeflich, Peter M. Haverty, Robert Soriano, William F. Forrest, Sherry Heldens, Huiyen Chen, Karen Toy, Connie Ha, Wei Zhou, Kyung Song, Lori S. Friedman, Lukas C. Amler, Garret M. Hampton, John Molliat, Marcia Belvin, and Mark R. Lackner

Triggering Fbw7-Mediated Proteasomal Degradation of c-Myc by Oridonin Induces Cell Growth Inhibition and Apoptosis
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miRNA-29b Suppresses Prostate Cancer Metastasis by Regulating Epithelial–Mesenchymal Transition Signaling

Peng Ru, Robert Steele, Philip Newhall, Nancy J. Phillips, Karoly Toth, and Ratna B. Ray

Therapeutic Significance of Estrogen Receptor β Agonists in Gliomas

Gangadhara R. Sareddy, Binoj C. Nair, Vijay K. Gonugunta, Quan-guang Zhang, Andrew Brenner, Darrell W. Brann, Rajeshwar Rao Tekmal, and Ratna K. Vadlamudi

Multiple Antigenic Peptides Based on H-2Kb–Restricted CTL Epitopes from Murine Heparanase Induce a Potent Antitumor Immune Response In Vivo

Xu-Dong Tang, Guo-Zhen Wang, Jun Guo, Mu-Han Liu, Chuan Li, Ning Li, Ya-Ling Chao, Chang-Zhu Li, Yu-Yun Wu, Chang-Jiang Hu, Dian-Chun Fang, and Shi-Ming Yang

Cotargeting MAPK and PI3K Signaling with Concurrent Radiotherapy as a Strategy for the Treatment of Pancreatic Cancer

Terence M. Williams, Athena R. Flecha, Paul Keller, Ashwin Ram, David Karnak, Stefanie Galbán, Craig J. Galbán, Brian D. Ross, Theodore S. Lawrence, Alnawaz Rehemtulla, and Judith Sebolt-Leopold

High TUBB3 Expression, an Independent Prognostic Marker in Patients with Early Non–Small Cell Lung Cancer Treated by Preoperative Chemotherapy, Is Regulated by K-Ras Signaling Pathway

Guénaëlle Levallet, Emmanuel Bergot, Martine Antoine, Christian Creveuil, Adriana O. Santos, Michelle Beau-Faller, Florence de Fraipont, Elisabeth Brambilla, Jérôme Levallet, Franck Morin, Virginie Westedt, Marie Wissez, Elisabeth Quoix, Didier Debieuvre, Fatéme Dubois, Isabelle Rouquette, Jean-Louis Pujol, Denis Moro-Sibilot, Jacques Camonis, Gérard Zalcman, on behalf of the Intergroupe Francophone de Cancérologie Thoracique (IFCT)

Retraction in Part: A Genomic Approach to Identify Molecular Pathways Associated with Chemotherapy Resistance

Correction: Narciclasine, a Plant Growth Modulator, Activates Rho and Stress Fibers in Glioblastoma Cells

Several allosteric MEK inhibitors are in clinical development and have been designed to treat patients with tumors harboring RAS/RAF pathway alterations. Acquired resistance to this class of inhibitors is a pressing clinical problem. To identify strategies to overcome this resistance, Hatzivassiliou and colleagues derived and characterized three independent MEK inhibitor-resistant cell lines. All of the resistant cell lines harbored mutations in the allosteric binding pocket of MEK that is targeted by arylamine MEK inhibitors. In all cases the MEK resistant cell lines retained their addiction to the MAPK pathway and remained sensitive to a selective inhibitor of the ERK1/2 kinases, suggesting a role for ERK inhibitors in combating or preventing MEK inhibitor resistance. For details, see article by Hatzivassiliou and colleagues on page 1143.