

REVIEWS

- 855 **Tumor Cell Death and ATP Release Prime Dendritic Cells and Efficient Anticancer Immunity**

Laetitia Aymeric, Lionel Apetoh, François Ghiringhelli, Antoine Tesniere, Isabelle Martins, Guido Kroemer, Mark J. Smyth, and Laurence Zitvogel

- 859 **Rethinking the Warburg Effect with Myc Micromanaging Glutamine Metabolism**

Chi V. Dang

PERSPECTIVES

- 863 **Vascular Endothelial Growth Factor—A Positive and Negative Regulator of Tumor Growth**

Laura M. Vecchiarelli-Federico, David Cervi, Mehran Haeri, Yanmei Li, Andras Nagy, and Yaacov Ben-David

PRIORITY REPORT

- 868 **Chemogenomic Profiling Provides Insights into the Limited Activity of Irreversible EGFR Inhibitors in Tumor Cells Expressing the T790M EGFR Resistance Mutation**

Martin L. Sos, Haridas B. Rode, Stefanie Heynck, Martin Peifer, Florian Fischer, Sabine Klüter, Vijaykumar G. Pawar, Cecile Reuter, Johannes M. Heuckmann, Jonathan Weiss, Lars Ruddigkeit, Matthias Rabiller, Mirjam Koker, Jeffrey R. Simard, Matthäus Getlik, Yuki Yuza, Tzu-Hsiu Chen, Heidi Greulich, Roman K. Thomas, and Daniel Rauh

CLINICAL STUDIES

- 875 **Phase I Trial of Intraperitoneal Administration of an Oncolytic Measles Virus Strain Engineered to Express Carcinoembryonic Antigen for Recurrent Ovarian Cancer**

Evanthia Galanis, Lynn C. Hartmann, William A. Cliby, Harry J. Long, Prema P. Peethambaram, Brigitte A. Barrette, Judith S. Kaur, Paul J. Haluska, Jr., Ileana Aderca, Paula J. Zollman, Jeff A. Sloan, Gary Keeney,

Pamela J. Atherton, Karl C. Podratz, Sean C. Dowdy, C. Robert Stanhope, Timothy O. Wilson, Mark J. Federspiel, Kah-Whye Peng, and Stephen J. Russell

INTEGRATED SYSTEMS AND TECHNOLOGIES

- 883 **Novel Candidate Cancer Genes Identified by a Large-Scale Cross-Species Comparative Oncogenomics Approach**

Jenny Mattison, Jaap Kool, Anthony G. Uren, Jeroen de Ridder, Lodewyk Wessels, Jos Jonkers, Graham R. Bignell, Adam Butler, Alistair G. Rust, Markus Brosch, Catherine H. Wilson, Louise van der Weyden, David A. Largaespada, Michael R. Stratton, P. Andy Futreal, Maarten van Lohuizen, Anton Berns, Lara S. Collier, Tim Hubbard, and David J. Adams

- 896 **Exon-Based Clustering of Murine Breast Tumor Transcriptomes Reveals Alternative Exons Whose Expression Is Associated with Metastasis**

Martin Dutertre, Magali Lacroix-Triki, Keltouma Driouch, Pierre de la Grange, Lise Gratadou, Samantha Beck, Stefania Millevoi, Jamal Tazi, Rosette Lidereau, Stephan Vagner, and Didier Auboeuf

MICROENVIRONMENT AND IMMUNOLOGY

- 906 **Efficacious Immune Therapy in Chronic Myelogenous Leukemia (CML) Recognizes Antigens That Are Expressed on CML Progenitor Cells**

Melinda A. Biernacki, Ovidiu Marina, Wandi Zhang, Fenglong Liu, Ingmar Bruns, Ann Cai, Donna Neuberg, Christine M. Canning, Edwin P. Alyea, Robert J. Soiffer, Vladimir Brusic, Jerome Ritz, and Catherine J. Wu

- 916 **NF- κ B, and not MYCN, Regulates MHC Class I and Endoplasmic Reticulum Aminopeptidases in Human Neuroblastoma Cells**

Matteo Forloni, Sonia Albini, Maria Zaira Limongi, Loredana Cifaldi, Renata Boldrini, Maria Rita Nicotra, Giuseppe Giannini, Pier Giorgio Natali, Patrizio Giacomini, and Doriana Fruci

925 **Effects of Acute versus Chronic Hypoxia on DNA Damage Responses and Genomic Instability**

Isabel M. Pires, Zuzana Bencokova, Manuela Milani, Lisa K. Folkes, Ji-Liang Li, Mike R. Stratford, Adrian L. Harris, and Ester M. Hammond

936 **Lung Metastasis Fails in MMTV-PyMT Oncomice Lacking S100A4 Due to a T-Cell Deficiency in Primary Tumors**

Birgitte Grum-Schwensen, Jörg Klingelhöfer, Mariam Grigorian, Kasper Almholt, Boye Schnack Nielsen, Eugene Lukanidin, and Noona Ambartsumian

948 **Variable Inhibition of Thrombospondin 1 against Liver and Lung Metastases through Differential Activation of Metalloproteinase ADAMTS1**

Yoon-Jin Lee, Moritz Koch, Daniel Karl, Antoni X. Torres-Collado, Namali T. Fernando, Courtney Rothrock, Darshini Kuruppu, Sandra Ryeom, M. Luisa Iruela-Arispe, and Sam S. Yoon

MOLECULAR AND CELLULAR PATHOBIOLOGY

957 **Regulation of Ceramide Synthase-Mediated Crypt Epithelium Apoptosis by DNA Damage Repair Enzymes**

Jimmy A. Rotolo, Judith Mesicek, Jerzy Maj, Jean-Philip Truman, Adriana Haimovitz-Friedman, Richard Kolesnick, and Zvi Fuks

968 **Smad Signaling Is Required to Maintain Epigenetic Silencing during Breast Cancer Progression**

Panagiotis Papageorgis, Arthur W. Lambert, Sait Ozturk, Fangming Gao, Hongjie Pan, Upender Manne, Yuriy O. Alekseyev, Arunthathi Thiagalingam, Hamid M. Abdolmaleky, Marc Lenburg, and Sam Thiagalingam

979 **SOX9 Elevation in the Prostate Promotes Proliferation and Cooperates with *PTEN* Loss to Drive Tumor Formation**

Martin K. Thomsen, Laurence Ambroisine, Sarah Wynn, Kathryn S.E. Cheah, Christopher S. Foster, Gabrielle Fisher, Daniel M. Berney, Henrik Møller, Victor E. Reuter, Peter Scardino, Jack Cuzick, Narasimhan Ragavan, Paras B. Singh, Francis L. Martin, Christopher M. Butler, Colin S. Cooper, and Amanda Swain on behalf of the Transatlantic Prostate Group

988 **Identification of Breast Tumor Mutations in *BRCA1* That Abolish Its Function in Homologous DNA Recombination**

Derek J.R. Ransburgh, Natsuko Chiba, Chikashi Ishioka, Amanda Ewart Toland, and Jeffrey D. Parvin

996 ***BRCA1* Represses Amphiregulin Gene Expression**

Ekaterina P. Lamber, Andrew A. Horwitz, and Jeffrey D. Parvin

PREVENTION AND EPIDEMIOLOGY

1006 **Inactivation of the Quinone Oxidoreductases *NQO1* and *NQO2* Strongly Elevates the Incidence and Multiplicity of Chemically Induced Skin Tumors**

Jun Shen, Roberto J. Barrios, and Anil K. Jaiswal

1015 **Pancreatic Cancer Risk and ABO Blood Group Alleles: Results from the Pancreatic Cancer Cohort Consortium**

Brian M. Wolpin, Peter Kraft, Myron Gross, Kathy Helzlsouer, H. Bas Bueno-de-Mesquita, Emily Stepnowski, Rachael Z. Stolzenberg-Solomon, Alan A. Arslan, Eric J. Jacobs, Andrea LaCroix, Gloria Petersen, Wei Zheng, Demetrius Albanes, Naomi E. Allen, Laufey Amundadottir, Garnet Anderson, Marie-Christine Boutron-Ruault, Julie E. Buring, Federico Canzian, Stephen J. Chanock, Sandra Clipp, John Michael Gaziano, Edward L. Giovannucci, Göran Hallmans, Susan E. Hankinson, Robert N. Hoover, David J. Hunter, Amy Hutchinson, Kevin Jacobs, Charles Kooperberg, Shannon M. Lynch, Julie B. Mendelsohn, Dominique S. Michaud, Kim Overvad, Alpa V. Patel, Aleksandar Rajkovic, Maria-José Sánchez, Xiao-Ou Shu, Nadia Slimani, Gilles Thomas, Geoffrey S. Tobias, Dimitrios Trichopoulos, Paolo Vineis, Jarmo Virtamo, Jean Wactawski-Wende, Kai Yu, Anne Zeleniuch-Jacquotte, Patricia Hartge, and Charles S. Fuchs

1024 **Definition of a Functional Single Nucleotide Polymorphism in the Cell Migration Inhibitory Gene *M1P* That Affects the Risk of Breast Cancer**

Fangfang Song, Ping Ji, Hong Zheng, Fengju Song, Yingmei Wang, Xishan Hao, Qingyi Wei, Wei Zhang, and Kexin Chen

- 1033 **Nek4 Status Differentially Alters Sensitivity to Distinct Microtubule Poisons**
Jason Doles and Michael T. Hemann
- 1042 **Resveratrol Promotes Autophagic Cell Death in Chronic Myelogenous Leukemia Cells via JNK-Mediated p62/SQSTM1 Expression and AMPK Activation**
Alexandre Puissant, Guillaume Robert, Nina Fenouille, Frederic Luciano, Jill-Patrice Cassuto, Sophie Raynaud, and Patrick Auberger
- 1053 **Sunitinib Acts Primarily on Tumor Endothelium rather than Tumor Cells to Inhibit the Growth of Renal Cell Carcinoma**
Dan Huang, Yan Ding, Yan Li, Wang-Mei Luo, Zhong-Fa Zhang, John Snider, Kristin VandenBeldt, Chao-Nan Qian, and Bin Tean Teh
- 1063 **Interleukin-8 Mediates Resistance to Antiangiogenic Agent Sunitinib in Renal Cell Carcinoma**
Dan Huang, Yan Ding, Ming Zhou, Brian I. Rini, David Petillo, Chao-Nan Qian, Richard Kahnoski, P. Andrew Futreal, Kyle A. Furge, and Bin Tean Teh
- 1072 **Interleukin-15 and Its Receptor Augment Dendritic Cell Vaccination against the *neu* Oncogene through the Induction of Antibodies Partially Independent of CD4 Help**
Jason C. Steel, Charmaine A. Ramlogan, Ping Yu, Yoshio Sakai, Guido Forni, Thomas A. Waldmann, and John C. Morris
- 1082 **A Flow Cytometry Method to Quantitate Internalized Immunotoxins Shows that Taxol Synergistically Increases Cellular Immunotoxins Uptake**
Yujian Zhang, Johanna K. Hansen, Laiman Xiang, Seiji Kawa, Masanori Onda, Mitchell Ho, Raffit Hassan, and Ira Pastan
- 1090 **FKBPL Regulates Estrogen Receptor Signaling and Determines Response to Endocrine Therapy**
Hayley D. McKeen, Christopher Byrne, Puthen V. Jithesh, Christopher Donley, Andrea Valentine, Anita Yakkundi, Martin O'Rourke, Charles Swanton, Helen O. McCarthy, David G. Hirst, and Tracy Robson

- 1101 **Multivalent DR5 Peptides Activate the TRAIL Death Pathway and Exert Tumoricidal Activity**
Valeria Pavet, Julien Beyrath, Christophe Pardin, Alexandre Morizot, Marie-Charlotte Lechner, Jean-Paul Briand, Miriam Wendland, Wolfgang Maison, Sylvie Fournel, Olivier Micheau, Gilles Guichard, and Hinrich Gronemeyer
- 1111 **Combined Treatment of Pancreatic Cancer with Mithramycin A and Tolfenamic Acid Promotes Sp1 Degradation and Synergistic Antitumor Activity**
Zhiliang Jia, Yong Gao, Liwei Wang, Qiang Li, Jun Zhang, Xiangdong Le, Daoyan Wei, James C. Yao, David Z. Chang, Suyun Huang, and Keping Xie
- 1120 **PERK-Dependent Regulation of Ceramide Synthase 6 and Thioredoxin Play a Key Role in *mda-7/IL-24*-Induced Killing of Primary Human Glioblastoma Multiforme Cells**
Adly Yacoub, Hossein A. Hamed, Jeremy Allegood, Clint Mitchell, Sarah Spiegel, Maciej S. Lesniak, Besim Ogretmen, Rupesh Dash, Devanand Sarkar, William C. Broaddus, Steven Grant, David T. Curiel, Paul B. Fisher, and Paul Dent

TUMOR AND STEM CELL BIOLOGY

- 1130 **Silencing of RON Receptor Signaling Promotes Apoptosis and Gemcitabine Sensitivity in Pancreatic Cancers**
Jocelyn Logan-Collins, Ryan M. Thomas, Peter Yu, Dawn Jaquish, Evangeline Mose, Randall French, William Stuart, Rebecca McClaine, Bruce Aronow, Robert M. Hoffman, Susan E. Waltz, and Andrew M. Lowy
- 1141 **The EphB6 Receptor Cooperates with c-Cbl to Regulate the Behavior of Breast Cancer Cells**
Luke Truitt, Tanya Freywald, John DeCoteau, Nigel Sharfe, and Andrew Freywald
- 1154 **EWS/FLI1 Oncogene Activates Caspase 3 Transcription and Triggers Apoptosis *In vivo***
Eun Jung Sohn, Hongjie Li, Karen Reidy, Lisa F. Beers, Barbara L. Christensen, and Sean Bong Lee
- 1164 **Isoform-Specific Phosphoinositide 3-Kinase Inhibitors Exert Distinct Effects in Solid Tumors**
Kyle A. Edgar, Jeffrey J. Wallin, Megan Berry, Leslie B. Lee, Wei Wei Prior, Deepak Sampath, Lori S. Friedman, and Marcia Belvin

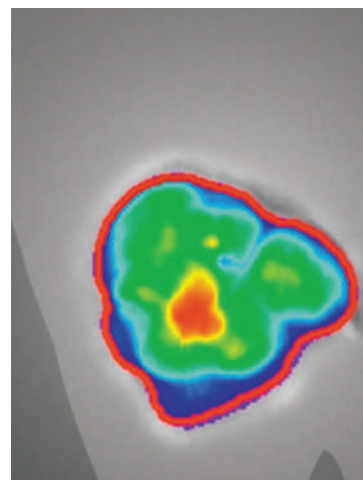
- 1173 **Caffeine-Mediated Inhibition of Calcium Release Channel Inositol 1,4,5-Trisphosphate Receptor Subtype 3 Blocks Glioblastoma Invasion and Extends Survival**
Sang Soo Kang, Kyung-Seok Han, Bo Mi Ku, Yeon Kyung Lee, Jinpyo Hong, Hye Young Shin, Antoine G. Almonte, Dong Ho Woo, Daniel J. Brat, Eun Mi Hwang, Seung Hyun Yoo, Chun Kee Chung, Sung-Hye Park, Sun Ha Paek, Eun Joo Roh, Sung Joong Lee, Jae-Yong Park, Stephen F. Traynelis, and C. Justin Lee
- 1184 **The G Protein–Coupled Receptor GPR30 Inhibits Proliferation of Estrogen Receptor–Positive Breast Cancer Cells**
Eric A. Ariazi, Eugen Brailoiu, Smitha Yerrum, Heather A. Shupp, Michael J. Slifker, Heather E. Cunliffe, Michael A. Black, Anne L. Donato, Jeffrey B. Arterburn, Tudor I. Oprea, Eric R. Prossnitz, Nae J. Dun, and V. Craig Jordan
- 1195 **Proapoptotic Kinase MST2 Coordinates Signaling Crosstalk between RASSF1A, Raf-1, and Akt**
David Romano, David Matallanas, Gregory Weitsman, Christian Preisinger, Tony Ng, and Walter Kolch
- 1204 **Heterotrimerization of the Growth Factor Receptors erbB2, erbB3, and Insulin-like Growth Factor-I Receptor in Breast Cancer Cells Resistant to Herceptin**
Xiaoping Huang, Lizhi Gao, Shuiliang Wang, James L. McManaman, Ann D. Thor, XiaoHe Yang, Francisco J. Esteva, and Bolin Liu
- 1215 **PSF1, a DNA Replication Factor Expressed Widely in Stem and Progenitor Cells, Drives Tumorigenic and Metastatic Properties**
Yumi Nagahama, Masaya Ueno, Satoru Miyamoto, Eiiichi Morii, Takashi Minami, Naoki Mochizuki, Hideyuki Saya, and Nobuyuki Takakura
- 1225 **Role of Cationic Channel TRPV2 in Promoting Prostate Cancer Migration and Progression to Androgen Resistance**
Michaël Monet, V'yacheslav Lehen'kyi, Florian Gackiere, Virginie Firlje, Matthieu Vandenberghe, Morad Roudbaraki, Dimitra Gkika, Albin Pourtier, Gabriel Bidaux, Christian Slomianny, Philippe Delcourt, François Rassendren, Jean-Pierre Bergerat, Jocelyn Ceraline, Florence Cabon, Sandrine Humez, and Natalia Prevarskaya
- 1236 **ZNF238 Is Expressed in Postmitotic Brain Cells and Inhibits Brain Tumor Growth**
Valérie M. Tatard, Chaomei Xiang, Jaclyn A. Biegel, and Nadia Dahmane
- 1247 **Bile Acid Reflux Contributes to Development of Esophageal Adenocarcinoma via Activation of Phosphatidylinositol-Specific Phospholipase C γ 2 and NADPH Oxidase NOX5-S**
Jie Hong, Jose Behar, Jack Wands, Murray Resnick, Li Juan Wang, Ronald A. DeLellis, David Lambeth, and Weibiao Cao
- 1256 **Evidence of Limited Contributions for Intratumoral Steroidogenesis in Prostate Cancer**
Johannes Hofland, Wytske M. van Weerden, Natasja F.J. Dits, Jacobie Steenberg, Geert J.L.H. van Leenders, Guido Jenster, Fritz H. Schröder, and Frank H. de Jong
- 1265 **RBCK1 Drives Breast Cancer Cell Proliferation by Promoting Transcription of Estrogen Receptor α and Cyclin B1**
Nina Gustafsson, Chunyan Zhao, Jan-Åke Gustafsson, and Karin Dahlman-Wright

CORRECTIONS

- 1275 **Correction: Antibody-Drug Conjugates for the Treatment of Non-Hodgkin's Lymphoma: Target and Linker-Drug Selection**
- 1275 **Correction: An A13 Repeat within the 3'-Untranslated Region of Epidermal Growth Factor Receptor (EGFR) Is Frequently Mutated in Microsatellite Instability Colon Cancers and Is Associated with Increased EGFR Expression**
- 1275 **Correction: Glioblastoma Cells Require Glutamate Dehydrogenase to Survive Impairments of Glucose Metabolism or Akt Signaling**

ABOUT THE COVER

The RON receptor tyrosine kinase is overexpressed in premalignant pancreatic intraepithelial neoplasia and in the majority of pancreatic cancers; however, the pathobiological significance of RON overexpression in pancreatic cancers has yet to be fully established. In this study, Logan-Collins and colleagues show that in both murine and human pancreatic cancer cells, RON signaling regulates the expression of genes implicated in cancer cell survival. shRNA-mediated silencing of RON in pancreatic cancer xenografts inhibited their growth, primarily by increasing susceptibility to apoptosis and by sensitizing them to gemcitabine treatment. In addition, the authors show that escape from RON silencing is associated with re-expression of RON and/or expression of phosphorylated forms of the related c-Met or epidermal growth factor receptors. Given these findings, the authors propose that RON signaling mediates cell survival and *in vivo* resistance to gemcitabine in pancreatic cancer, and they reveal mechanisms through which pancreatic cancer cells may circumvent RON-directed therapies. For details, see the article by Logan-Collins and colleagues on page 1130 of this issue.



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70 (3)

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