

Highlights of This Issue 1187

SPECIAL FEATURES

CCR Translations

- 1189 **PET Imaging of Tumor Growth: Not as Easy as It Looks**
Anthony F. Shields
See article p. 1303
- 1192 **Rehabilitation for Oncogene Addiction: Role of Immunity in Cellular Sobriety**
David L. Bajor and Robert H. Vonderheide
See article p. 1386

CCR New Strategies

- 1195 **New Strategies in Melanoma: Molecular Testing in Advanced Disease**
Scott E. Woodman, Alexander J. Lazar, Kenneth D. Aldape, and Michael A. Davies

Molecular Pathways

- 1201 **Molecular Pathways: Beta-Adrenergic Signaling in Cancer**
Steven W. Cole and Anil K. Sood
- 1207 **Molecular Pathways: Hypoxia Response in Immune Cells Fighting or Promoting Cancer**
Asis Palazón, Julián Aragonés, Aizea Morales-Kastresana, Manuel Ortiz de Landázuri, and Ignacio Melero

Review

- 1214 ***Here, There Be Dragons: Charting Autophagy-Related Alterations in Human Tumors***
Chandra B. Lebovitz, Svetlana B. Bortnik, and Sharon M. Gorski

HUMAN CANCER BIOLOGY

- 1227 **Activation of PI3K Signaling in Merkel Cell Carcinoma**
Valentina Nardi, Youngchul Song, Juan A. Santamaria-Barria, Arjola K. Cosper, Quynh Lam, Anthony C. Faber, Genevieve M. Boland, Beow Y. Yeap, Kristin Bergethon, Vanessa L. Scialabba, Hensin Tsao, Jeffrey Settleman, David P. Ryan, Darrell R. Borger, Atul K. Bhan, Mai P. Hoang, Anthony J. Iafrate, James C. Cusack, Jeffrey A. Engelman, and Dora Dias-Santagata
- 1237 **Infiltration of Lynch Colorectal Cancers by Activated Immune Cells Associates with Early Staging of the Primary Tumor and Absence of Lymph Node Metastases**
Noel F.C.C. de Miranda, Danny Goudkade, Ekaterina S. Jordanova, Carli M.J. Tops, Frederik J. Hes, Hans F.A. Vasen, Tom van Wezel, and Hans Morreau
- 1246 **HDL of Patients with Type 2 Diabetes Mellitus Elevates the Capability of Promoting Breast Cancer Metastasis**
Bing Pan, Hui Ren, Yubin He, Xiaofeng Lv, Yijing Ma, Jing Li, Li Huang, Baoqi Yu, Jian Kong, Chenguang Niu, Youyi Zhang, Wen-bing Sun, and Lemin Zheng
- 1257 **Downregulation of SMG-1 in HPV-Positive Head and Neck Squamous Cell Carcinoma Due to Promoter Hypermethylation Correlates with Improved Survival**
Evgenia Gubanova, Brandee Brown, Sergei V. Ivanov, Thomas Helleday, Gordon B. Mills, Wendell G. Yarbrough, and Natalia Issaeva

CANCER THERAPY: PRECLINICAL

- 1268 **Telomestatin Impairs Glioma Stem Cell Survival and Growth through the Disruption of Telomeric G-Quadruplex and Inhibition of the Proto-oncogene, *c-Myb***
Takeshi Miyazaki, Yang Pan, Kaushal Joshi, Deepti Purohit, Bin Hu, Habibe Demir, Sarmistha Mazumder, Sachiko Okabe, Takao Yamori, Mariano Viapiano, Kazuo Shin-ya, Hiroyuki Seimiya, and Ichiro Nakano

1281 **SKI-606, an Src Inhibitor, Reduces Tumor Growth, Invasion, and Distant Metastasis in a Mouse Model of Thyroid Cancer**
Won Gu Kim, Celine J. Guigon, Laura Fozzatti, Jeong Won Park, Changxue Lu, Mark C. Willingham, and Sheue-yann Cheng

1291 **Polymeric Nanoparticle-Encapsulated Hedgehog Pathway Inhibitor HPI-1 (NanoHHI) Inhibits Systemic Metastases in an Orthotopic Model of Human Hepatocellular Carcinoma**
Yang Xu, Venugopal Chenna, Chaoxin Hu, Hai-Xiang Sun, Mehtab Khan, Haibo Bai, Xin-Rong Yang, Qing-Feng Zhu, Yun-Fan Sun, Anirban Maitra, Jia Fan, and Robert A. Anders

IMAGING, DIAGNOSIS, PROGNOSIS

1303 **[¹⁸F]FLT-PET Imaging Does Not Always "Light Up" Proliferating Tumor Cells**
Cathy C. Zhang, Zhengming Yan, Wenlin Li, Kyle Kuszpit, Cory L. Painter, Qin Zhang, Patrick B. Lappin, Tim Nichols, Maruja E. Lira, Timothy Affolter, Neeta R. Fahey, Carleen Cullinane, Mary Spilker, Kenneth Zasadny, Peter O'Brien, Dana Buckman, Anthony Wong, and James G. Christensen
See commentary p. 1189

1313 **Epidermal Growth Factor Receptor Protein Detection in Head and Neck Cancer Patients: A Many-Faceted Picture**
Juliette Thariat, Marie-Christine Etienne-Grimaldi, Dominique Grall, René-Jean Bensadoun, Anne Cayre, Frédérique Penault-Llorca, Laurence Veracini, Mireille Francoual, Jean-Louis Formento, Olivier Dassonville, Dominique De Raucourt, Lionel Geoffrois, Philippe Giraud, Séverine Racadot, Sylvain Morinière, Gérard Milano, and Ellen Van Obberghen-Schilling

1323 **Combination of a Novel Gene Expression Signature with a Clinical Nomogram Improves the Prediction of Survival in High-Risk Bladder Cancer**
Markus Riester, Jennifer M. Taylor, Andrew Feifer, Theresa Koppie, Jonathan E. Rosenberg, Robert J. Downey, Bernard H. Bochner, and Franziska Michor

1334 **Copy Number Losses Define Subgroups of Dedifferentiated Liposarcoma with Poor Prognosis and Genomic Instability**
Aimee M. Crago, Nicholas D. Socci, Penelope DeCarolis, Rachael O'Connor, Barry S. Taylor, Li-Xuan Qin, Cristina R. Antonescu, and Samuel Singer

1341 **Elucidating Prognosis and Biology of Breast Cancer Arising in Young Women Using Gene Expression Profiling**
Hatem A. Azim Jr, Stefan Michiels, Philippe L. Bedard, Sandeep K. Singhal, Carmen Crisciello, Michail Ignatiadis, Benjamin Haibe-Kains, Martine J. Piccart, Christos Sotiriou, and Sherene Loi

1352 **Integrative Survival-Based Molecular Profiling of Human Pancreatic Cancer**
Timothy R. Donahue, Linh M. Tran, Reginald Hill, Yunfeng Li, Anne Kovochich, Joseph H. Calvopina, Sanjeet G. Patel, Nanping Wu, Antreas Hindoyan, James J. Farrell, Xinmin Li, David W. Dawson, and Hong Wu

1364 **Human Prostate Cancer in a Clinically Relevant Xenograft Mouse Model: Identification of $\beta(1,6)$ -Branched Oligosaccharides as a Marker of Tumor Progression**
Tobias Lange, Sebastian Ullrich, Imke Müller, Michael F. Nentwich, Katrin Stübke, Susanne Feldhaus, Christine Knies, Olaf J.C. Hellwinkel, Robert L. Vessella, Claudia Abramjuk, Mario Anders, Jennifer Schröder-Schwarz, Thorsten Schlomm, Hartwig Huland, Guido Sauter, and Udo Schumacher

1374 **High-Risk Ovarian Cancer Based on 126-Gene Expression Signature Is Uniquely Characterized by Downregulation of Antigen Presentation Pathway**
Kosuke Yoshihara, Tatsuhiko Tsunoda, Daichi Shigemizu, Hiroyuki Fujiwara, Masayuki Hatae, Hisaya Fujiwara, Hideaki Masuzaki, Hidetaka Katabuchi, Yosuke Kawakami, Aikou Okamoto, Takayoshi Nogawa, Noriomi Matsumura, Yasuhiro Udagawa, Tsuyoshi Saito, Hiroaki Itamochi, Masashi Takano, Etsuko Miyagi, Tamotsu Sudo, Kimio Ushijima, Haruko Iwase, Hiroyuki Seki, Yasuhisa Terao, Takayuki Enomoto, Mikio Mikami, Kohei Akazawa, Hitoshi Tsuda, Takuya Moriya, Atsushi Tajima, Ituro Inoue, and Kenichi Tanaka for The Japanese Serous Ovarian Cancer Study Group

CANCER THERAPY: CLINICAL

- 1386 **Selective BRAF Inhibitors Induce Marked T-cell Infiltration into Human Metastatic Melanoma**
James S. Wilmott, Georgina V. Long, Julie R. Howle, Lauren E. Haydu, Raghwa N. Sharma, John F. Thompson, Richard F. Kefford, Peter Hersey, and Richard A. Scolyer
See commentary p. 1192
- 1395 **Results of a Phase 1 Study of AME-133v (LY2469298), an Fc-Engineered Humanized Monoclonal Anti-CD20 Antibody, in FcγRIIIa-Genotyped Patients with Previously Treated Follicular Lymphoma**
Andres Forero-Torres, Sven de Vos, Brad L. Pohlman, Maksim Pashkevich, Damien M. Cronier, Nam H. Dang, Susan P. Carpenter, Barrett W. Allan, James G. Nelson, Christopher A. Slapak, Mitchell R. Smith, Brian K. Link, James E. Wooldridge, and Kristen N. Ganjoo
- 1404 **Prospective Trial of Synchronous Bevacizumab, Erlotinib, and Concurrent Chemoradiation in Locally Advanced Head and Neck Cancer**
David S. Yoo, John P. Kirkpatrick, Oana Craciunescu, Gloria Broadwater, Bercedis L. Peterson, Madeline D. Carroll, Robert Clough, James R. MacFall, Jenny Hoang, Richard L. Scher, Ramon M. Esclamado, Frank R. Dunphy, Neal E. Ready, and David M. Brizel
- 1415 **Phase I Clinical and Pharmacokinetic Evaluation of the Vascular-Disrupting Agent OXi4503 in Patients with Advanced Solid Tumors**
Dan M. Patterson, Martin Zweifel, Mark R. Middleton, Patricia M. Price, Lisa K. Folkes, Michael R.L. Stratford, Phil Ross, Sarah Halford, Jane Peters, Jai Balkissoon, Dai J. Chaplin, Anwar R. Padhani, and Gordon J.S. Rustin
- 1426 **Lenalidomide-Induced Immunomodulation in Multiple Myeloma: Impact on Vaccines and Antitumor Responses**
Kimberly Noonan, Lakshmi Rudraraju, Anna Ferguson, Amy Emerling, Marcela F. Pasetti, Carol A. Huff, and Ivan Borrello
- 1435 **A Phase II Study of Gefitinib for Aggressive Cutaneous Squamous Cell Carcinoma of the Head and Neck**
Carol M. Lewis, Bonnie S. Glisson, Lei Feng, Fiona Wan, Ximing Tang, Ignacio I. Wistuba, Adel K. El-Naggar, David I. Rosenthal, Mark S. Chambers, Robert A. Lustig, and Randal S. Weber

PREDICTIVE BIOMARKERS AND PERSONALIZED MEDICINE

- 1447 **CD26 Overexpression Is Associated with Prolonged Survival and Enhanced Chemosensitivity in Malignant Pleural Mesothelioma**
Keisuke Aoe, Vishwa Jeet Amatya, Nobukazu Fujimoto, Kei Ohnuma, Osamu Hosono, Akio Hiraki, Masanori Fujii, Taketo Yamada, Nam H. Dang, Yukio Takeshima, Kouki Inai, Takumi Kishimoto, and Chikao Morimoto
- 1457 **Sunitinib Therapy for Melanoma Patients with *KIT* Mutations**
David R. Minor, Mohammed Kashani-Sabet, Maria Garrido, Steven J. O'Day, Omid Hamid, and Boris C. Bastian
- 1464 **Pathway-Specific Analysis of Gene Expression Data Identifies the PI3K/Akt Pathway as a Novel Therapeutic Target in Cervical Cancer**
Julie K. Schwarz, Jacqueline E. Payton, Ramachandran Rashmi, Tao Xiang, Yunhe Jia, Phyllis Huettner, Buck E. Rogers, Qin Yang, Mark Watson, Janet S. Rader, and Perry W. Grigsby
- 1472 **Mechanisms of Resistance to Crizotinib in Patients with *ALK* Gene Rearranged Non-Small Cell Lung Cancer**
Robert C. Doebele, Amanda B. Pilling, Dara L. Aisner, Tatiana G. Kutateladze, Anh T. Le, Andrew J. Weickhardt, Kimi L. Kondo, Derek J. Linderman, Lynn E. Heasley, Wilbur A. Franklin, Marileila Varela-Garcia, and D. Ross Camidge

CORRECTIONS

- 1483 **Correction: Noninvasive Detection of Breast Cancer Lymph Node Metastasis Using Carbonic Anhydrases IX and XII Targeted Imaging Probes**
- 1484 **Correction: Glutamatergic Pathway Targeting in Melanoma: Single-Agent and Combinatorial Therapies**

ABOUT THE COVER

Following treatment with a G-quadruplex ligand, telomestatin, glioma stem cells rapidly developed punctate nuclear 53BP1 foci. Of note, some of these foci colocalized with nontelomeric DNA, thereby representing both telomeric and nontelomeric dysfunction-induced foci, a hallmark of deprotected DNA damage. The loss of tumor stemness is likely associated with a failure in the DNA damage response elicited by telomestatin in glioma stem cells. For details, see the article by Miyazaki and colleagues on page 1268 of this issue.



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