

BREAKING INSIGHTS

- 6527** Highlights from Recent Cancer Literature


REVIEW

- 6529** Tolerance of Chromosomal Instability in Cancer: Mechanisms and Therapeutic Opportunities
Eva Gronroos and Carlos López-García


CANCER RESEARCH HIGHLIGHTS

- 6536** Cancer Immunity and Gene Expression Data: A Quick Tool for Immunophenotype Evaluation
Masayuki Hirano
See related article, p. 6575

PRIORITY REPORT

- 6539** Pathologic Oxidation of PTPN12 Underlies ABL1 Phosphorylation in Hereditary Leiomyomatosis and Renal Cell Carcinoma
 Yang Xu, Paul Taylor, Joshua Andrade, Beatrix Ueberheide, Brian Shuch, Peter M. Glazer, Ranjit S. Bindra, Michael F. Moran, W. Marston Linehan, and Benjamin G. Neel
Significance: This work identifies a novel mechanism of activation of the oncogenic kinase ABL1 via ROS-induced, oxidation-mediated inactivation of cognate protein tyrosine phosphatases.

MOLECULAR CELL BIOLOGY


- 6549** DNA Polymerase Eta Prevents Tumor Cell-Cycle Arrest and Cell Death during Recovery from Replication Stress
 Ryan P. Barnes, Wei-Chung Tsao, George-Lucian Moldovan, and Kristin A. Eckert
Significance: This study demonstrates that replication stress upregulates Pol η (POLH) in tumor cells and reveals a role for Pol η in tumor cell recovery following replication stress.

- 6561** CDK1 Interacts with Sox2 and Promotes Tumor Initiation in Human Melanoma
Dinoop Ravindran Menon, Yuchun Luo, John J. Arcaroli, Sucai Liu, Lekha Nair KrishnanKutty, Douglas G. Osborne, Yang Li, Jenny Mae Samson, Stacey Bagby, Aik-Choon Tan, William A. Robinson, Wells A. Messersmith, and Mayumi Fujita

Significance: These findings uncover CDK1 as a new regulator of Sox2 during tumor initiation and implicate the CDK1-Sox2 interaction as a potential therapeutic target in cancer.

TUMOR BIOLOGY AND IMMUNOLOGY

- 6575** TIP: A Web Server for Resolving Tumor Immunophenotype Profiling
Liwen Xu, Chunyu Deng, Bo Pang, Xinxin Zhang, Wei Liu, Gaoming Liao, Huating Yuan, Peng Cheng, Feng Li, Zhilin Long, Min Yan, Tingting Zhao, Yun Xiao, and Xia Li
Significance: TIP is a one-stop shop platform that can help biologists, clinicians, and researchers conveniently evaluate anticancer immune activity with their own gene expression data.
See related commentary, p. 6536

- 6581** Reduced CD160 Expression Contributes to Impaired NK-cell Function and Poor Clinical Outcomes in Patients with HCC
 Haoyu Sun, Jing Xu, Qiang Huang, Mei Huang, Kun Li, Kun Qu, Hao Wen, Renyong Lin, Meijuan Zheng, Haiming Wei, Weihua Xiao, Rui Sun, Zhigang Tian, and Cheng Sun
Significance: These findings show that reduced number and function of CD160⁺ NK cells in the tumor microenvironment contribute to immune escape of HCC; blocking TGFβ1 restores IFNγ production of CD160⁺ NK cells.


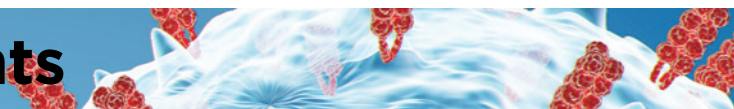
- 6594** CDK8 Selectively Promotes the Growth of Colon Cancer Metastases in the Liver by Regulating Gene Expression of TIMP3 and Matrix Metalloproteinases
 Jiaxin Liang, Mengqian Chen, Daniel Hughes, Alexander A. Chumanevich, Serena Altiglia, Vimala Kaza, Chang-Uk Lim, Hippokratis Kiaris, Karthikeyan Mythreye, Maria Marjorette Pena, Eugenia V. Broude, and Igor B. Roninson
Significance: These findings demonstrate that inhibition of the transcription-regulating kinase CDK8 exerts a site-specific tumor-suppressive effect on colon cancer growth in the liver, representing a unique therapeutic opportunity for the treatment of advanced colon cancer.

Table of Contents



- 6607** LPA4-Mediated Vascular Network Formation Increases the Efficacy of Anti-PD-1 Therapy against Brain Tumors
Daisuke Eino, Yohei Tsukada, Hisamichi Naito, Yonehiro Kanemura, Tomohiro Iba, Taku Wakabayashi, Fumitaka Muramatsu, Hiroyasu Kidoya, Hideyuki Arita, Naoki Kagawa, Yasunori Fujimoto, Kazuhiro Takara, Haruhiko Kishima, and Nobuyuki Takakura
Significance: These findings demonstrate that lysophosphatidic acid, a lipid mediator, promotes development of a fine capillary network in brain tumors by inducing tightening of endothelial cell-to-cell adhesion, facilitating improved drug delivery and lymphocyte penetration.
- 6621** Nedd4-Binding Protein 1 and TNFAIP3-Interacting Protein 1 Control MHC-1 Display in Neuroblastoma
Lotte Spel, Joppe Nieuwenhuis, Rianne Haarsma, Elmer Stickel, Onno B. Bleijerveld, Maarten Altelaar, Jaap Jan Boelens, Thijn R. Brummelkamp, Stefan Nierkens, and Marianne Boes
Significance: Aberrant regulation of NF- κ B and MHC-1 in neuroblastoma tumors provides new targets for immunotherapeutic approaches against neuroblastoma.
- 6632** Circulating Glioma Cells Exhibit Stem Cell-like Properties
Tianrun Liu, Haineng Xu, Menggui Huang, Wenjuan Ma, Deeksha Saxena, Robert A. Lustig, Michelle Alonso-Basanta, Zhenfeng Zhang, Donald M. O'Rourke, Lin Zhang, Yanqing Gong, Gary D. Kao, Jay F. Dorsey, and Yi Fan
Significance: These findings identify CTCs as an alternative source for in situ tumor invasion and recurrence through local micrometastasis, warranting eradication of systemic "out-of-tumor" CTCs as a promising new therapeutic opportunity for GBM.
- 6643** Intratumoral Immunotherapy with XCL1 and sFlt3L Encoded in Recombinant Semliki Forest Virus-Derived Vectors Fosters Dendritic Cell-Mediated T-cell Cross-Priming
Alfonso R. Sánchez-Paulete, Álvaro Teijeira, José I. Quetglas, María E. Rodríguez-Ruiz, Álvaro Sánchez-Arráez, Sara Labiano, Iñaki Etxeberria, Arantza Azpilikueta, Elixabet Bolaños, María Cristina Ballesteros-Briones, Noelia Casares, Sergio A. Quezada, Pedro Berraondo, David Sancho, Cristian Smerdou, and Ignacio Melero
Significance: These findings demonstrate that transgenic expression of sFLT3L and XCL1 in tumor cells mediates cross-priming of, and elicits potent antitumor activity from, CD8 T lymphocytes, particularly in combination with CD4 T-cell depletion.

- 6655** Pomalidomide Inhibits PD-L1 Induction to Promote Antitumor Immunity
Yuki Fujiwara, Yi Sun, Robert J. Torphy, Jiadai He, Katsuhiko Yanaga, Barish H. Edil, Richard D. Schulick, and Yuwen Zhu
Significance: These findings report that the immunomodulatory drug pomalidomide, widely used to treat myeloma and other cancers, enhances antitumor immunity by inhibiting PD-1/PD-L1 expression.
- 6666** A p53-Responsive miRNA Network Promotes Cancer Cell Quiescence
Ting La, Guang Zhi Liu, Margaret Farrelly, Nicole Cole, Yu Chen Feng, Yuan Yuan Zhang, Simonne K. Sherwin, Hamed Yari, Hessam Tabatabaee, Xu Guang Yan, Su Tang Guo, Tao Liu, Rick F. Thorne, Lei Jin, and Xu Dong Zhang
Significance: Two novel p53-responsive microRNAs whose distinct mechanisms of action both stabilize p27 to promote cell quiescence and may serve as therapeutic avenues for improving outcomes of cancer treatment.


TRANSLATIONAL SCIENCE

- 6680** The TLR7/8/9 Antagonist IMO-8503 Inhibits Cancer-Induced Cachexia
Federica Calore, Priya Londhe, Paolo Fadda, Giovanni Nigita, Lucia Casadei, Gioacchino Paolo Marceca, Matteo Fassan, Francesca Lovat, Pierluigi Gasparini, Lara Rizzotto, Nicola Zanesi, Devine Jackson, Svasti Mehta, Patrick Nana-Sinkam, Deepa Sampath, Raphael E. Pollock, Denis C. Guttridge, and Carlo M. Croce
Significance: Cancer-associated cachexia is a significant problem for patients with cancer that remain poorly understood, understudied, and inadequately treated; these findings report a potential new therapeutic for the treatment of TLR7-mediated cancer cachexia.
- 6691** Pharmacological Inhibition of PARP6 Triggers Multipolar Spindle Formation and Elicits Therapeutic Effects in Breast Cancer
Zebin Wang, Shaun E. Grosskurth, Tony Cheung, Philip Petteruti, Jingwen Zhang, Xin Wang, Wenxian Wang, Farzin Gharahdaghi, Jiaquan Wu, Nancy Su, Ryan T. Howard, Michele Mayo, Dan Widzowski, David A. Scott, Jeffrey W. Johannes, Michelle L. Lamb, Deborah Lawson, Jonathan R. Dry, Paul D. Lyne, Edward W. Tate, Michael Zinda, Keith Mikule, Stephen E. Fawell, Corinne Reimer, and Huawei Chen
Significance: These findings describe a new inhibitor of PARP6 and identify a novel function of PARP6 in regulating activation of Chk1 in breast cancer cells.

Table of Contents

CONVERGENCE AND TECHNOLOGIES

- 6703** A Convolutional Neural Network Uses Microscopic Images to Differentiate between Mouse and Human Cell Lines and Their Radioresistant Clones
Masayasu Toratani, Masamitsu Konno, Ayumu Asai, Jun Koseki, Koichi Kawamoto, Keisuke Tamari, Zhihao Li, Daisuke Sakai, Toshihiro Kudo, Taroh Satoh, Katsutoshi Sato, Daisuke Motooka, Daisuke Okuzaki, Yuichiro Doki, Masaki Mori, Kazuhiko Ogawa, and Hideshi Ishii
Significance: This study demonstrates rapid and accurate identification of radioresistant tumor cells in culture using artificial intelligence; this should have applications in future preclinical cancer research.

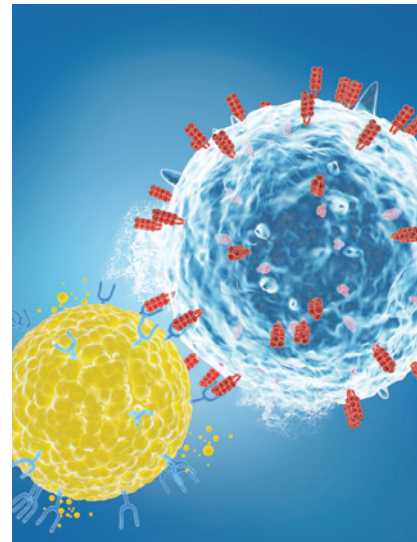
 AC icon indicates Author Choice
For more information please visit www.aacrjournals.org

RETRACTIONS

- 6708** Retraction: Interleukin-12 Deficiency Is Permissive for Angiogenesis in UV Radiation-Induced Skin Tumors
Syed M. Meeran, Suchitra Katiyar, Craig A. Elmets, and Santosh K. Katiyar
- 6709** Retraction: (-)-Epigallocatechin-3-Gallate Prevents Photocarcinogenesis in Mice through Interleukin-12-Dependent DNA Repair
Syed M. Meeran, Sudheer K. Mantena, Craig A. Elmets, and Santosh K. Katiyar

ABOUT THE COVER

Reduced number and impaired function of CD160⁺ natural killer (NK) cells in the tumor microenvironment contributes to the immune escape of human hepatocellular carcinoma. Patients with fewer CD160⁺ NK cells within tumors exhibit worse disease condition and higher recurrence rate. High level of TGFβ1 interferes with the production of IFNγ by CD160⁺ NK cells, the blocking of which specifically restores IFNγ production of CD160⁺ NK cells to normal levels. Restoring the expression of CD160 and blocking TGFβ1 appear to be a promising therapeutic strategy against liver cancer. For details, see article by Sun and colleagues on page 6581.



Cancer Research

The Journal of Cancer Research (1916–1930) | The American Journal of Cancer (1931–1940)

78 (23)

Cancer Res 2018;78:6527-6709.

Updated version Access the most recent version of this article at:
<http://cancerres.aacrjournals.org/content/78/23>

E-mail alerts [Sign up to receive free email-alerts](#) related to this article or journal.

Reprints and Subscriptions To order reprints of this article or to subscribe to the journal, contact the AACR Publications Department at pubs@aacr.org.

Permissions To request permission to re-use all or part of this article, use this link <http://cancerres.aacrjournals.org/content/78/23>.
Click on "Request Permissions" which will take you to the Copyright Clearance Center's (CCC) Rightslink site.