Cancer Research
Table of Contents
December 1, 2015 • Volume 75 • Number 23

BREAKING ADVANCES

4999 Highlights from Recent Cancer Literature

REVIEWS

5001 p53: Protection against Tumor Growth beyond Effects on Cell Cycle and Apoptosis
Xuyi Wang, Evan R. Simpson, and Kristy A. Brown

5008 Antibody-Dependent Phagocytosis of Tumor Cells by Macrophages: A Potent Effector Mechanism of Monoclonal Antibody Therapy of Cancer
Nuray Gül and Marjolein van Egmond

5014 Mechanisms of Cancer Cell Dormancy—Another Hallmark of Cancer?
Albert C. Yeh and Sridhar Ramaswamy

PRIORITY REPORT

5023 Fluorophore-NanoLuc BRET Reporters Enable Sensitive In Vivo Optical Imaging and Flow Cytometry for Monitoring Tumorigenesis
Franz X. Schaub, Md. Shamim Reza, Colin A. Flaveny, Weimim Li, Adele M. Musicant, Sany Hodha, Min Guo, John L. Cleveland, and Antonio L. Amelio
Précis: This study describes an extremely bright luciferase reporter that can enable highly sensitive, yet inexpensive methods for in vivo monitoring of small numbers of tumor cells, such as found at early metastatic sites, with greatly reduced image acquisition times.

5058 Fibulin-5 Blocks Microenvironmental ROS in Pancreatic Cancer
Miao Wang, Mary Topalowski, Jason E. Toombs, Christopher M. Wright, Zachary R. Moore, David A. Boothman, Hiromi Yanagisawa, Huamin Wang, Agnieszka Witsiekicz, Diego H. Castrillon, and Rolf A. Brekken
Précis: These findings reveal an integrin-based mechanism that attenuates ROS production and promotes cancer progression, with implications for a novel general strategy to reprogram the tumor microenvironment to improve therapeutic response.

5070 The CUL4B/AKT/β-Catenin Axis Restricts the Accumulation of Myeloid-Derived Suppressor Cells to Prohibit the Establishment of a Tumor-Permissive Microenvironment
Yanyan Qian, Jupeng Yuan, Huili Hu, Qi Feng Yang, Jisheng Li, Shuqian Zhang, Baichun Jiang, Changshun Shao, and Yaqin Gong
Précis: These surprising findings describe a previously uncharacterized antitumorigenic role for CUL4B in the hematopoietic system, where it restricts the accumulation and activity of myeloid-derived suppressor cells to prevent the establishment of a tumor permissive microenvironment, underscoring mechanisms by which immunosurveillance may be compromised by certain therapeutic strategies.

MICROENVIRONMENT AND IMMUNOLOGY

5034 Chemotherapy Induces Programmed Cell Death-Ligand 1 Overexpression via the Nuclear Factor-κB to Foster an Immunosuppressive Tumor Microenvironment in Ovarian Cancer
Jin Peng, Junzo Hamanishi, Noriomi Matsumura, Kaoru Abiko, Kumuruz Murat, Tsukasa Baba, Ken Yamaguchi, Naoki Horikawa, Yuko Horoe, Susan X. Murphy, Ikuo Konishi, and Masaki Mandai
Précis: These findings that chemotherapeutic agents induce immunosuppression by activating NF-κB/PD-L1 signaling in ovarian cancer suggest that the antitumor response may be enhanced through combination strategies encompassing both chemotherapy and immunotherapy.

5046 Obesity Contributes to Ovarian Cancer Metastatic Success through Increased Lipogenesis, Enhanced Vascularity, and Decreased Infiltration of M1 Macrophages
Précis: These findings highlight the potential mechanisms by which obesity contributes to ovarian cancer metastatic success, with important implications for patient outcomes.

5058 Obesity Contributes to Ovarian Cancer Metastatic Success through Increased Lipogenesis, Enhanced Vascularity, and Decreased Infiltration of M1 Macrophages
Précis: These findings highlight the potential mechanisms by which obesity contributes to ovarian cancer metastatic success, with important implications for patient outcomes.

December 1, 2015 • Volume 75 • Number 23

Cancer Research
Table of Contents

5046 Obesity Contributes to Ovarian Cancer Metastatic Success through Increased Lipogenesis, Enhanced Vascularity, and Decreased Infiltration of M1 Macrophages
Précis: These findings highlight the potential mechanisms by which obesity contributes to ovarian cancer metastatic success, with important implications for patient outcomes.

5058 Obesity Contributes to Ovarian Cancer Metastatic Success through Increased Lipogenesis, Enhanced Vascularity, and Decreased Infiltration of M1 Macrophages
Précis: These findings highlight the potential mechanisms by which obesity contributes to ovarian cancer metastatic success, with important implications for patient outcomes.

5070 The CUL4B/AKT/β-Catenin Axis Restricts the Accumulation of Myeloid-Derived Suppressor Cells to Prohibit the Establishment of a Tumor-Permissive Microenvironment
Yanyan Qian, Jupeng Yuan, Huili Hu, Qi Feng Yang, Jisheng Li, Shuqian Zhang, Baichun Jiang, Changshun Shao, and Yaqin Gong
Précis: These surprising findings describe a previously uncharacterized antitumorigenic role for CUL4B in the hematopoietic system, where it restricts the accumulation and activity of myeloid-derived suppressor cells to prevent the establishment of a tumor permissive microenvironment, underscoring mechanisms by which immunosurveillance may be compromised by certain therapeutic strategies.
Table of Contents

5084 Serum Immunoregulatory Proteins as Predictors of Overall Survival of Metastatic Melanoma Patients Treated with Ipilimumab
Précis: These findings define the immunomodulatory factors CXCL11 and sMICA as predictive markers in melanoma patients least likely to benefit from treatment with the checkpoint inhibitor ipilimumab.

MOLECULAR AND CELLULAR PATHOBIOLOGY

5093 Checkpoint Kinase 2 Negatively Regulates Androgen Sensitivity and Prostate Cancer Cell Growth
Huy Q. Ta, Melissa L. Ivey, Henry F. Frierson Jr, Mark R. Conaway, Jaroslaw Dziegielewski, James M. Larner, and Daniel Gioeli
Précis: This study illuminates a DNA damage response pathway that intersects with the G2–M cell-cycle checkpoint to influence the development of castration-resistant prostate cancer, with potential implications for its treatment.

THERAPEUTICS, TARGETS, AND CHEMICAL BIOLOGY

5106 Generation of a Selective Small Molecule Inhibitor of the CBF/p300 Bromodomain for Leukemia Therapy
Sarah Picaud, Oleg Fedorov, Angeliki Thanasopoulou, Katharina Leonards, Katherine Jones, Julia Meier, Heidi Olzscha, Octovia Monteiro, Sarah Martin, Martin Philpott, Anthony Tumber, Panagis Filipakopoulos, Clarence Yapp, Christopher Wells, Ka Hing Che, Andrew Bannister, Samuel Robson, Umesh Kumar, Nigel Parr, Kevin Lee, Dave Iugo, Philip Jeffrey, Simon Taylor, Matteo L. Yecellio, Chas Bourne, Paul E. Brennan, Alison O’Mahony, Sharlene Velichko, Susanne Müller, Duncan Hay, Danette L. Daniels, Marjeta Urb, Nicholas B. La Thangue, Tony Kouzourides, Rab Prinjha, Jürg Schwaller, and Stefan Knapp
Précis: These findings highlight a mechanistically potent strategy to inhibit a histone acetyltransferase that impairs self-renewal of leukemic cells, with implications to improve current treatment approaches for aggressive leukemias.

5120 Hyperthermia Selectively Targets Human Papillomavirus in Cervical Tumors via p53-Dependent Apoptosis
Arlene L. Oel, Caspar M. van Leeuwen, Rosemarie ten Cate, Hans M. Rodermond, Marrije R. Buist, Lukas J.A. Stalpers, Johannes Crezee, H. Petra Kok, Jan Paul Medema, and Nicolaas A.P. Franken
Précis: These findings reveal mechanistic insights underlying the response of HPV-positive cervical cancers to hyperthermia therapy, with immediate implications for patient outcomes.

5130 Naturally Occurring Isothiocyanates Exert Anticancer Effects by Inhibiting Deubiquitinating Enzymes
Ann P. Lawson, Marcus J.C. Long, Rory T. Coffey, Yu Qian, Eranthie Weerapana, Fadil E. Oualid, and Lizbeth Hedstrom
Précis: This study offers a novel unifying mechanism to understand the cancer-fighting properties of a class of natural compounds found in broccoli and other cruciferous vegetables that might help fight a variety of diseases characterized by inflammatory pathology.

TUMOR AND STEM CELL BIOLOGY

5143 WDR5 Supports an N-Myc Transcriptional Complex That Drives a Protumorigenic Gene Expression Signature in Neuroblastoma
Yuting Sun, Jessica L. Bell, Daniel Carter, Samuele Gherardi, Rebecca C. Poulos, Giorgio Milazzo, Jason W.H. Wong, Rima Al-Awar, Andrew E. Tee, Pei Y. Liu, Bing Liu, Bernard Atmadibrata, Matthew Wong, Toby Trahair, Quan Zhao, Jason M. Shohet, Ygal Haupt, Johannes H. Schulte, Peter J. Brown, Cheryl H. Arrowsmith, Masoud Vedadi, Karen L. MacKenzie, Giovanni Perini, Glenn M Marshall, Antony Brathwaite, and Tao Liu
Précis: These results identify the histone methylation regulator WDR5 as a key cofactor for N-Myc-driven transcriptional activation and tumorigenesis, offering evidence of its candidacy as a novel therapeutic target for MYCN-amplified neuroblastomas.
ATDC/TRIM29 Drives Invasive Bladder Cancer Formation through miRNA-Mediated and Epigenetic Mechanisms
Phillip L. Palmbos, Lidong Wang, Huibin Yang, Yin Wang, Jacob Leflein, McKenzie L. Ahmet, John E. Wilkinson, Chandan Kumar-Sinha, Gina M. Ney, Scott A. Tomlins, Stephanie Daignault, Lakshmi P. Kunju, Xue-Ru Wu, Yair Lotan, Monica Liebert, Mats E. Ljungman, and Diane M. Simeone
Précis: Identification of a novel oncogenic driver of bladder carcinogenesis introduces a candidate biomarker and therapeutic target in a setting that has not kept pace with progress made in other cancers.

Correction: CD38 in Hairy Cell Leukemia Is a Marker of Poor Prognosis and a New Target for Therapy

ABOUT THE COVER
CBP/p300 is functionally implicated in the progression of multiple hematological malignancies. Picaud and colleagues developed a new selective and highly potent chemical probe I-CBP112 targeting the bromodomains of CBP/p300. Treatment of human acute myeloid leukemia (AML) cells growing in methylcellulose with 1, 3, and 5 μmol/L of the compound reduced the clonogenic growth of the cells in a dose-dependent manner. Cytospin preparations of I-BP112–treated primary AML blasts showed morphologic signs of differentiation. For details, see article by Picaud and colleagues on page 5106.
75 (23)

Cancer Res 2015;75:4999-5167.

Updated version
Access the most recent version of this article at:
http://cancerres.aacrjournals.org/content/75/23

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, contact the AACR Publications Department at permissions@aacr.org.