**BREAKING ADVANCES**

617 Highlights from Recent Cancer Literature

**REVIEWS**

619 Emerging Links between E2F Control and Mitochondrial Function
Elizaveta V. Benevolenskaya and Maxim V. Frolov

624 GOLPH3 Links the Golgi, DNA Damage, and Cancer
Matthew D. Buschman, Juliati Rahajeng, and Seth J. Field

**PRIORITY REPORT**

628 A Unique Subset of Epithelial Ovarian Cancers with Platinum Sensitivity and PARP Inhibitor Resistance
Raphael Ceccaldi, Kevin W. O’Connor, Kent W. Mouw, Adam Y. Li, Ursula A. Matulonis, Alan D. D’Andrea, and Panagiotis A. Konstantinopoulos

Précis: These findings reveal a mechanism of platinum sensitivity that alters sensitivity to PARP inhibitors in a discordant manner, with potential implications for trials of this new class of drugs in ovarian cancer.

**MICROENVIRONMENT AND IMMUNOLOGY**

635 Accelerated Tumor Progression in Mice Lacking the ATP Receptor P2X7
Elena Adinolfi, Marina Capace, Alessia Franceschini, Simonetta Falzoni, Anna L. Giuliani, Alessandra Rottondo, Alba C. Sarti, Massimo Bonora, Susanne Syberg, Domenica Corigliano, Paolo Pinton, Niklas R. Jorgensen, Luigi Abelli, Laura Emionite, Lizzia Raffaghello, Vito Pistoia, and Francesco Di Virgilio

Précis: These provocative genetic results challenge the notion that inflammation solely promotes tumor growth by showing how deletion of an important proinflammatory receptor, the P2X/ATP receptor, can impair antitumor responses and promote malignant progression.

**MOLECULAR AND CELLULAR PATHOBIOLOGY**

666 Genetic Mutation of p53 and Suppression of the miR-17–92 Cluster Are Synthetic Lethal in Non–Small Cell Lung Cancer due to Upregulation of Vitamin D Signaling

Précis: These genetic findings suggest that vitamin D receptor agonists may be highly efficacious in p53 mutant lung cancers, a possibility with immediate implications for clinical evaluation.
Stem Cell Transplantation Reverses Chemotherapy-Induced Cognitive Dysfunction

Précis: Cancer survivors suffer from impaired cognition due to chemotherapy, a condition informally referred to as “chemobrain,” but this very common side effect in cancer survivors has been little studied as an unmet medical need.

687 eIF4E Threshold Levels Differ in Governing Normal and Neoplastic Expansion of Mammary Stem and Luminal Progenitor Cells
Svetlana Avdulov, Jeremy Herrera, Karen Smith, Mark Peterson, Jose R. Gomez-Garcia, Thomas C. Bradnell, Kathryn L. Schwertfeger, Alexey O. Benyumov, I. Carlos Manivel, Shunan Li, Anja-Katrin Bielinsky, Douglas Yee, Peter B. Bitterman, and Vitaly A. Polunovsky

Précis: eIF4E overexpression, which occurs widely in cancer, appears to enable cells to evade DNA damage checkpoints, a feature that is associated with threshold levels but not changes in RNA cap-binding capabilities as might have been suspected.

698 Interaction between p53 Mutation and a Somatic HDMX Biomarker Better Defines Metastatic Potential in Breast Cancer

Précis: This study develops a simple paired biomarker for the p53 pathway in breast cancer, rendering it more clinically useful for predicting metastatic progression and patient prognosis.

709 HOXB7 Promotes Malignant Progression by Activating the TGFβ Signaling Pathway
Shou Liu, Rodeok Jin, Yvonne Hui, Jie Fu, Chunfa Jie, Sheng Feng, David Reisman, Qian Wang, Daping Fan, Saraswati Sukumar, and Hexin Chen

Précis: These findings reveal a mechanism that is required not only to promote cancer cell invasion and migration, but also to recruit and activate immunosuppressive tumor-associated macrophages in the tumor microenvironment.

720 Reprogramming of the ERα and ERβ Target Gene Landscape Triggers Tamoxifen Resistance in Breast Cancer
Verena Tewes, Ronald Simon, Petra Schroeter, Magdalena Schlott, Tobias Anzeneder, Reinhard Büttner, Vladimir Benes, Guido Sauter, Barbara Burwinkel, Robert I. Nicholson, Hans-Peter Sinn, Andreas Schnevewiss, Ulrich Deuschle, Marc Zapata, Stefanie Heck, and Peter Lichter

Précis: These findings suggest that activation of the estrogen-related receptor ERα promotes resistance to antiendocrine therapy in breast cancer and provide a rationale to explore this receptor as a drug target for mitigating the endocrine-resistant phenotype in patients.

732 BRCA2-Deficient Sarcomatoid Mammary Tumors Exhibit Multidrug Resistance
Janneke E. Jaspers, Wendy Sol, Ariena Kersbergen, Andreas Schlicker, Charlotte Guyader, Guotai Xu, Lodewyk Wessels, Piet Borst, Jos Jonkers, and Sven Rottenberg

Précis: Epithelial-to-mesenchymal transition in murine tumors is associated with an acquisition of multidrug resistance due to increased expression of genes encoding drug efflux transporters.

TUMOR AND STEM CELL BIOLOGY

742 ERK5 Is a Critical Mediator of Inflammation-Driven Cancer
Katherine C. Finegan, Diana Perez-Madrigal, James R. Hitchin, Clare C. Davies, Allan M. Jordan, and Cathy Tournier

Précis: These findings highlight a kinase that fosters chronic inflammation in the setting of carcinogenesis, a key issue in understanding how an inflamed microenvironment supports cancer progression.

754 Epigenetic Silencing of miR-490-3p Reactivates the Chromatin Remodeler SMARCD1 to Promote Helicobacter pylori–Induced Gastric Carcinogenesis

Précis: This study shows how miRNA misregulation of a member of the SWI/SNF chromatin remodeling family contributes to the development of infection-associated stomach cancers.
Suppressing TGFβ Signaling in Regenerating Epithelia in an Inflammatory Microenvironment Is Sufficient to Cause Invasive Intestinal Cancer
Hiroko Oshima, Mizuho Nakayama, Tae-Su Han, Kuniko Naoi, Xiaoli Ju, Yusuke Maeda, Sylvie Robine, Kiichiro Tsuchiya, Toshiro Sato, Hiroshi Sato, Makoto Mark Takeo, and Masanobu Oshima
Précis: These provocative results show how invasive colon cancers can develop simply as a result of chronic inflammation that engenders evolution of immune escape, alongside epithelial cell regeneration that seeks to restore colonic tissue in the face of ongoing inflammation.

LETTER TO THE EDITOR
Cep63 Recruits Cdk1 to the Centrosome—Letter
Mohammad Alsara, Harald Löffler, Anne Fechter, Jiri Bartek, and Alwin Krämer

CORRECTION
Correction: AIMP3 Haploinsufficiency Disrupts Oncogene-Induced p53 Activation and Genomic Stability

ABOUT THE COVER
Human neural stem cell (hNSC) transplantation reverses chemotherapy-induced cognitive dysfunction through a mechanism involving the preservation of host neuronal morphology. The image shows Golgi-Cox impregnated neurons in the hippocampus of rats treated with chronic cyclophosphamide and engrafted with hNSCs. Disruptions to overall granule and CA1 pyramidal cell neuronal architecture caused by cyclophosphamide were ameliorated in the brains of rats receiving hNSC transplantation when analyzed 2 months posttransplantation. For further details, see article by Acharya and colleagues on page 676.