

GUEST EDITORIAL

- 393 | **Highlighting the Science of Cancer Prevention**
Elizabeth H. Blackburn

EDITORIAL COMMENTARY

- 394 | **Unprecedented Opportunities and Promise for Cancer Prevention Research**
Elizabeth H. Blackburn, Thea D. Tlsty, and Scott M. Lippman

PERSPECTIVES

- 403 | **NF- κ B Fans the Flames of Lung Carcinogenesis**
Kwok-Kin Wong, Tyler Jacks, and Glenn Dranoff
See article p. 424
- 406 | **The Ultimate in Cancer Chemoprevention: Cancer Vaccines**
Mary L. Disis
See article p. 438
- 410 | **Vaccine Prevention of Cancer: Can Endogenous Antigens Be Targeted?**
Louis M. Weiner, Rishi Surana, and Joseph Murray
See article p. 438
- 416 | **Interphase Cytogenetics of Sputum Cells for the Early Detection of Lung Carcinogenesis**
Sheila A. Prindiville and Thomas Ried
See article p. 447
- 420 | **Lung Cancer Biomarkers: FISHing in the Sputum for Risk Assessment and Early Detection**
Brigitte N. Gomperts, Avrum Spira, David E. Elashoff, and Steven M. Dubinett
See article p. 447

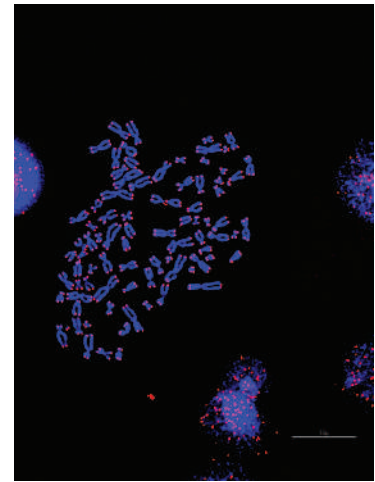
RESEARCH ARTICLES

- 424 | **Knockout of the Tumor Suppressor Gene *Gprc5a* in Mice Leads to NF- κ B Activation in Airway Epithelium and Promotes Lung Inflammation and Tumorigenesis**
Jiong Deng, Junya Fujimoto, Xiao-Feng Ye, Tao-Yan Men, Carolyn S. Van Pelt, Yu-Long Chen, Xiao-Feng Lin, Humam Kadara, Qingguo Tao, Dafna Lotan, and Reuben Lotan
See perspective p. 403
- 438 | **Vaccine against MUC1 Antigen Expressed in Inflammatory Bowel Disease and Cancer Lessens Colonic Inflammation and Prevents Progression to Colitis-Associated Colon Cancer**
Pamela L. Beatty, Sowmya Narayanan, Jean Gariépy, Sarangarajan Ranganathan, and Olivera J. Finn
See perspectives pp. 406 and 410
- 447 | **The Detection of Chromosomal Aneusomy by Fluorescence *In situ* Hybridization in Sputum Predicts Lung Cancer Incidence**
Marileila Varella-Garcia, Aline P. Schulte, Holly J. Wolf, William J. Feser, Chan Zeng, Sarah Braudrick, Xiang Yin, Fred R. Hirsch, Timothy C. Kennedy, Robert L. Keith, Anna E. Barón, Steven A. Belinsky, York E. Miller, Tim Byers, and Wilbur A. Franklin
See perspectives pp. 416 and 420
- 454 | **5-Deoxykaempferol Plays a Potential Therapeutic Role by Targeting Multiple Signaling Pathways in Skin Cancer**
Kyung Mi Lee, Ki Won Lee, Sanguine Byun, Sung Keun Jung, Sang Kwon Seo, Yong-Seok Heo, Ann M. Bode, Hyong Joo Lee, and Zigang Dong
- 466 | **Weight Loss via Exercise with Controlled Dietary Intake May Affect Phospholipid Profile for Cancer Prevention in Murine Skin Tissues**
Ping Ouyang, Yu Jiang, Hieu M. Doan, Linglin Xie, David Vasquez, Ruth Welti, Xiaoyu Su, Nanyan Lu, Betty Herndon, Shie-Shien Yang, Richard Jeannotte, and Weiqun Wang

- 478 **Serum Oxidized Protein and Prostate Cancer Risk within the Prostate Cancer Prevention Trial**
Ashrafal Hoque, Christine B. Ambrosone, Cathee Till, Phyllis J. Goodman, Cathy Tangen, Alan Kristal, Scott Lucia, Qiao Wang, Maya Kappil, Ian Thompson, Ann W. Hsing, Howard Parnes, and Regina M. Santella
- 484 **Sulforaphane Inhibits Constitutive and Interleukin-6–Induced Activation of Signal Transducer and Activator of Transcription 3 in Prostate Cancer Cells**
Eun-Ryeong Hahm and Shivendra V. Singh
- 495 **STAT2 Contributes to Promotion of Colorectal and Skin Carcinogenesis**
Ana M. Camero, Matthew R. Young, Roycelynn Mentor-Marcel, Gerd Bobe, Anthony J. Scarzello, Jennifer Wise, and Nancy H. Colburn
- 505 **Energy Balance, the PI3K-AKT-mTOR Pathway Genes, and the Risk of Bladder Cancer**
Jie Lin, Jianming Wang, Anthony J. Greisinger, H. Barton Grossman, Michele R. Forman, Colin P. Dinney, Ernest T. Hawk, and Xifeng Wu
- 518 **Nano-Bio-Chip Sensor Platform for Examination of Oral Exfoliative Cytology**
Shannon E. Weigum, Pierre N. Floriano, Spencer W. Redding, Chih-Ko Yeh, Stephen D. Westbrook, H. Stan McGuff, Alan Lin, Frank R. Miller, Fred Villarreal, Stephanie D. Rowan, Nadarajah Vigneswaran, Michelle D. Williams, and John T. McDevitt
- 529 **Fiberoptic Resonance Raman Spectroscopy to Measure Carotenoid Oxidative Breakdown in Live Tissues**
Brandon G. Bentz, Jason Diaz, Terry A. Ring, Mark Wade, Konrad Kennington, David M. Burnett, Robert McClane, and Frank A. Fitzpatrick
- 539 **The Core Circadian Gene *Cryptochrome 2* Influences Breast Cancer Risk, Possibly by Mediating Hormone Signaling**
Aaron E. Hoffman, Tongzhang Zheng, Chun-Hui Yi, Richard G. Stevens, Yue Ba, Yawei Zhang, Derek Leaderer, Theodore Holford, Johnni Hansen, and Yong Zhu
- 549 **Resveratrol Suppresses Colitis and Colon Cancer Associated with Colitis**
Xiangli Cui, Yu Jin, Anne B. Hofseth, Edsel Pena, Joshua Habiger, Alexander Chumanevich, Deepak Poudyal, Mitzi Nagarkatti, Prakash S. Nagarkatti, Udai P. Singh, and Lorne J. Hofseth
- 560 **Sulindac Metabolites Induce Proteosomal and Lysosomal Degradation of the Epidermal Growth Factor Receptor**
Heather A. Pangburn, Dennis J. Ahnen, and Pamela L. Rice

ABOUT THE COVER

Cultured melanoma cells were treated with colcemid to arrest growth at metaphase, made to swell with a hypotonic solution, fixed with methanol/acetic acid, and dropped on microscope slides to burst them and spread the chromosomes (blue) tipped by telomeres (red) shown in the center area of the cover image (approximately 3000× magnification; courtesy of Brad Stohr and Elizabeth Blackburn). Several nuclei did not burst during slide preparation (also displaying blue chromosomal DNA and red telomeres) and appear adjacent to the spread chromosomes. Chromosomal DNA was stained with 4',6-diamidino-2-phenylindole (DAPI), and telomeres were marked via a fluorescently labeled peptide nucleic-acid probe specific for telomeric DNA repeats. Telomere maintenance (and thus genomic instability) is sustained by the enzyme telomerase in normal cells. Short telomeres are associated with major cancer risk factors including smoking, inflammation, and obesity. Recently, chronic psychological stress, a risk factor for cancer in animal models, also has been shown to take its toll on telomere maintenance in humans. Therefore, the biology of telomeres and telomerase is a potential source of risk and drug-sensitivity markers for integrative, interdisciplinary cancer prevention involving behavioral and basic scientists, epidemiologists, clinical trialists, and biostatisticians. This biology reflects the exciting science of cancer prevention that enables advances in the practice and clinical impact of cancer prevention on several fronts. See articles by Blackburn, Tlsty, and Lippman (beginning on page 394) and Blackburn (on page 393) for more information.



Cancer Prevention Research

3 (4)

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