Metformin and Cancer Stem Cells: Old Drug, New Targets
Filip Bednar and Diane M. Simeone
Perspective on Bao et al., p. 355

Metformin Inhibits Cell Proliferation, Migration and Invasion by Attenuating CSC Function Mediated by Deregulating miRNAs in Pancreatic Cancer Cells
See Perspective on p. 351

Genetic Variants Associated with the Risk of Chronic Obstructive Pulmonary Disease with and without Lung Cancer
Mariza de Andrade, Yan Li, Randolph S. Marks, Claude Deschamps, Paul D. Scanlon, Curtis L. Olswold, Zhifu Sun, Julie M. Cunningham, David E. Midthun, and Ping Yang

Epigenetic Differences in Normal Colon Mucosa of Cancer Patients Suggest Altered Dietary Metabolic Pathways
Matthew L. Silviera, Brian P. Smith, Jasmine Powell, and Carmen Sapienza

A Dietary Pattern Associated with LINE-1 Methylation Alters the Risk of Developing Cervical Intraepithelial Neoplasia

Effect of 2-Month Controlled Green Tea Intervention on Lipoprotein Cholesterol, Glucose, and Hormone Levels in Healthy Postmenopausal Women
Anna H. Wu, Darcy Spicer, Frank Z. Stanczyk, Chi-Chen Tseng, Chung S. Yang, and Malcolm C. Pike

Functional Protein Pathway Activation Mapping of the Progression of Normal Skin to Squamous Cell Carcinoma

Effects of Energy Restriction and Wheel Running on Mammary Carcinogenesis and Host Systemic Factors in a Rat Model
Zongjian Zhu, Wei Qin Jiang, Jarrod H. Zacher, Elizabeth S. Neil, John N. McGinley, and Henry J. Thompson

Risk Factors for Malignant Melanoma in White and Non-White/Non–African American Populations: The Multiethnic Cohort
Sungshim Lani Park, Loic Le Marchand, Lynne R. Wilkens, Laurence N. Kolonel, Brian E. Henderson, Zuo-Feng Zhang, and Veronica Wendy Setiawan

Uterine Serous Carcinoma: Increased Familial Risk for Lynch-Associated Malignancies

Immunomodulation of Curcumin on Adoptive Therapy with T Cell Functional Imaging in Mice
Ya-Fang Chang, Hui-Yen Chuang, Chien-Hui Hsu, Ren-Shyan Liu, Sanjiv Sam Gambhir, and Jeng-Jong Hwang

Smoking Attenuates Transforming Growth Factor-β-Mediated Tumor Suppression Function through Downregulation of Smad3 in Lung Cancer
Debangshu Samanta, Adriana L. Gonzalez, Nagaraj Nagathihalli, Fei Ye, David P. Carbone, and Pran K. Datta
<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>464</td>
<td>DNA Methylation of Phosphatase and Actin Regulator 3 Detects Colorectal Cancer in Stool and Complements FIT</td>
<td>Bosch, Oort, Neerincx, Khalid-de Bakker, Terhaar sive Droste, Melotte, Jonkers, Masclee, Mongera, Grooteclaes, van Engeland, Carvalho, Meijer</td>
</tr>
<tr>
<td>473</td>
<td>Didymin Induces Apoptosis by Inhibiting N-Myc and Upregulating RKIP in Neuroblastoma</td>
<td>Singhal, Dalasanur Nagaprashantha, Vatsyayan, Awasthi, Singhal</td>
</tr>
<tr>
<td>484</td>
<td>ALDH1A1 Is a Novel EZH2 Target Gene in Epithelial Ovarian Cancer Identified by Genome-Wide Approaches</td>
<td>Li, Bitler, Vathipadiekal, Maradeo, Slifker, Creasy, Tummino, Cairns, Birrer, Zhang</td>
</tr>
</tbody>
</table>

**ABOUT THE COVER**

Anchorage-independent growth in semisolid medium and the formation of xenografts in immunocompromised mice are generally considered to be informative for assessing human cell tumorigenicity. Long-term treatment with cigarette smoke condensate (CSC) significantly increases (versus DMSO control or no treatment) the anchorage-independent growth of A549 lung adenocarcinoma cells. A549 cells were treated with CSC for 300 days (mimicking long-term cigarette smoking) and were allowed to grow for 14 days in soft agarose. The cover features a phase-contrast micropictogram (40× magnification) of colonies of these cells that do not require a solid substrate for growth, an important characteristic feature of cancer cells. 300-Day CSC treated cells were injected s.c. into athymic nude mice, producing tumors of significantly increased volume (P < 0.001) and rate of development (P < 0.01) at 12 weeks [versus injected 300-day DMSO or parental (no-treatment) cells, not shown]. These oncogenic effects were due partly to down-regulation of Smad3. Immortalized bronchial epithelial HPL1A cells, however, did not exhibit similar phenotypes, putatively because these cells may require a longer period of CSC treatment to undergo the additional genetic or epigenetic changes necessary to become tumorigenic. See article by Samanta et al. (beginning on page 453) for more information.
**Cancer Prevention Research**

5 (3)


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