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Dual PI3K/mTOR Inhibitors: Does p53 Modulate Response? Oleksandr Ekshyyan, Arunkumar Anandharaj, and Cherie-Ann O. Nathan
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CCR Perspectives in Drug Approval

Use of Multiple Endpoints and Approval Paths Depicts a Decade of FDA Oncology Drug Approvals Michael B. Shea, Samantha A. Roberts, Jessica C. Walrath, Jeff D. Allen, and Ellen V. Sigal

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Molecular Pathways: Environmental Estrogens Activate Nongenomic Signaling to Developmentally Reprogram the Epigenome Rebecca Lee Yean Wong and Cheryl Lyn Walker

Molecular Pathways: PI3K Pathway Targets in Triple-Negative Breast Cancers Valerrie Gordon and Shantanu Banerji

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S100B Promotes Glioma Growth through Chemotraction of Myeloid-Derived Macrophages Huaqing Wang, Leying Zhang, Ian Y. Zhang, Xuebo Chen, Anna Da Fonseca, Shilihua Wu, Hui Ren, Sam Badie, Sam Sadeghi, Mao Ouyang, Charles D. Warden, and Behnam Badie

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LETTER TO THE EDITOR

PD-L1 Expression in B-cell Lymphomas and Virus-Associated Malignancies—Letter

Mads Hald Andersen

Correction: Concomitant BRAF and PI3K/mTOR Blockade Is Required for Effective Treatment of BRAFV600E Colorectal Cancer

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ABOUT THE COVER

β-catenin is a transmembrane protein that associates with junctional proteins and assists with the maintenance of cell attachment. As revealed through immunofluorescent staining, β-catenin (shown in green) localizes to the cell membranes and within the lateral junctional complex in normal appendix tissue. In contrast, tissue samples from patients with pseudomyxoma peritonei display primarily cytoplasmic staining of β-catenin and virtually no staining at the intercellular boundaries. However, antibiotic treatment of patients with pseudomyxoma peritonei results in a significant increase in β-catenin within the cell membranes, appearing to aid in the renormalization of β-catenin distribution. For details, see the article by Semino-Mora and colleagues on page 3966 of this issue.