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The State of Molecular Biomarkers for the Early Detection of Lung Cancer
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Effect of Long-term Propranolol Treatment on Hepatocellular Carcinoma Incidence in Patients with HCV-Associated Cirrhosis
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Impact of Screening Test Performance and Cost on Mortality Reduction and Cost-effectiveness of Multimodal Ovarian Cancer Screening
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Targeting FACT Complex Suppresses Mammary Tumorigenesis in Her2/neu Transgenic Mice
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Lack of ABCG2 Shortens Latency of BRCA1-Deficient Mammary Tumors and This Is Not Affected by Genistein or Resveratrol

Alveolar Hypoxia Promotes Murine Lung Tumor Growth through a VEGFR-2/EGFR-Dependent Mechanism
Vijaya Karoor, Myfan Le, Daniel Merrick, Karen A. Fagan, Edward C. Dempsey, and York E. Miller

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

ABOUT THE COVER

Nuclear chromatin structure differs dramatically between normal, precancerous, and tumor cells and is still one of the most accurate markers for cancer diagnosis. Unfortunately, the nature of chromatin structure differences between normal and tumor cells is not well defined and there have been only limited attempts to exploit these differences for cancer prevention and therapy. Nevertheless, the demonstrated anticancer efficacy of HDAC inhibitors suggests that chromatin is likely a promising source of anticancer targets. A novel class of chromatin modifying small molecules, Curaxins, with broad anticancer activity in multiple models of cancer, were recently discovered. Curaxins inhibit activity of Facilitates Chromatin Transcription (FACT) complex. FACT is involved in chromatin remodeling in tumor and stem cells and is a promising candidate marker and target of cancer. Inhibition of FACT is accompanied by changes in chromatin structure as well as activity of several cancer-related transcriptional factors, which require FACT assistance for the transcription of their target genes. The cover features an immunofluorescence photomicrograph of mammary adenocarcinoma cells treated with Curaxin-137 and stained with antibodies to NF-κB, a well-known tumor-promoting transcriptional factor. Curaxin-137 causes nuclear accumulation of inactive NF-κB which is incapable of induction of transcription in the absence of FACT. See the article by Koman et al. (beginning on page 1025) for more details about the tumor-preventive activity of Curaxin-137.