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Mechanistic Contribution of Ubiquitous 15-Lipoxygenase-1 Expression Loss in Cancer Cells to Terminal Cell Differentiation Evasion
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Revisit of Field Cancerization in Squamous Cell Carcinoma of Upper Aerodigestive Tract: Better Risk Assessment with Epigenetic Markers
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Changes in Breast Density and Circulating Estrogens in Postmenopausal Women Receiving Adjuvant Anastrozole

Dietary Energy Balance Modulates Prostate Cancer Progression in Hi-Myc Mice
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Chemoprevention of Colon and Small Intestinal Tumorigenesis in APCMin/þ Mice by Licofelone, a Novel Dual 5-LOX/COX Inhibitor: Potential Implications for Human Colon Cancer Prevention
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Nonsteroidal Anti-inflammatory Drugs and Glioma in the NIH-AARP Diet and Health Study Cohort
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Possible Role of Visfatin in Hepatoma Progression and the Effects of Branched-Chain Amino Acids on Visfatin-Induced Proliferation in Human Hepatoma Cells
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Unfolded Protein Response Signaling and MAP Kinase Pathways Underlie Pathogenesis of Arsenic-Induced Cutaneous Inflammation
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Bitter Melon Extract Impairs Prostate Cancer Cell-Cycle Progression and Delays Prostatic Intraepithelial Neoplasia in TRAMP Model
Peng Ru, Robert Steele, Pratibha V. Nerurkar, Nancy Phillips, and Ratna B. Ray

Acknowledgment to Reviewers
ABOUT THE COVER

The cover image is a photomicrograph (200X magnification) of mouse skin stained with antibody to activating transcription factor 6 alpha (ATF6α; red). Nuclei were counterstained with 4',6-diamidino-2-phenylindole (DAPI; blue). The color images were taken separately using an Olympus BX 51 Fluorescent microscope and then merged. When unfolded protein response (UPR) is activated, ATF6α translocates from endoplasmic reticulum (ER) membrane to the Golgi apparatus, where it undergoes cleavage by site-1 protease (S1P) and S2P. Cleaved ATF6α migrates to the nucleus (violet, reflecting its overlay with DAPI blue staining) and induces transcription of UPR target genes. New work reported in this issue of the journal found that sub-chronic arsenic exposure activated reactive oxygen species (ROS)-dependent UPR signaling pathways (including the ATF6α pathway), which enhanced inflammation in murine skin. UPR signaling is under intensive investigation in inflammatory diseases and cancers. See article by Li et al. (beginning on page 2101) for more information.