GENETIC DRIVER EVENTS IN PREMALIGNANCY: LOH VALIDATED FOR MARKING THE RISK OF ORAL CANCER
Webster K. Cavenee
Perspective on Zhang et al., p. 1081

VALIDATION OF LOH PROFILES FOR ASSESSING ORAL CANCER RISK
Mark W. Lingen and Eva Szabo
Perspective on Zhang et al., p. 1081

THE GREAT ESCAPE: MICROBIOTAL LPS TAKES A TOLL ON THE LIVER
David S. Weiss
Perspective on Lin et al., p. 1090

LOSS OF HETEROZYGOSITY (LOH) PROFILES—VALIDATED RISK PREDICTORS FOR PROGRESSION TO ORAL CANCER
Lewei Zhang, Catherine F. Poh, Michele Williams, Denise M. Laronde, Ken Berean, Pamela J. Gardner, Huijun Jiang, Lang Wu, J. Jack Lee, and Miriam P. Rosin
See Perspectives on p. 1073 and p. 1075

GUT-DERIVED LIPOLYSACCHARIDE PROMOTES T-CELL–MEDIATED HEPATITIS IN MICE THROUGH TOLL-LIKE RECEPTOR 4
Yan Lin, Le-Xing Yu, He-Xin Yan, Wen Yang, Liang Tang, Hui-Lu Zhang, Qiong Liu, Shan-Shan Zou, Ya-Qin He, Chao Wang, Meng-Chao Wu, and Hong-Yang Wang
See Perspective on p. 1078

TAXIFOLIN SUPPRESSES UV-INDUCED SKIN CARCINOGENESIS BY TARGETING EGFR AND PI3K
Naomi Oi, Hanyong Chen, Myoung Ok Kim, Ronald A. Lubet, Ann M. Bode, and Zigang Dong

A CLINICAL RISK PREDICTION MODEL FOR BARRETT ESOPHAGUS
Aaron P. Thrift, Bradley J. Kendall, Nirmala Pandeya, Thomas L. Vaughan, David C. Whiteman, for the Study of Digestive Health

FAILURE RATES IN THE HEPATOCELLULAR CARCINOMA SURVEILLANCE PROCESS
Amir G. Singal, Adam C. Yopp, Samir Gupta, Celette Sugg Skinner, Ethan A. Halm, Eucharia Okolo, Mahendra Nehra, William M. Lee, Jorge A. Marrero, and Jasmin A. Tiro

AN EMLIN1-NEGATIVE MICROENVIRONMENT PROMOTES TUMOR CELL PROLIFERATION AND LYMPH NODE INVASION
Carla Danussi, Alessandra Petrucco, Bruna Wassermann, Teresa Maria Elisa Modica, Eliana Pivetta, Lisa Del Bel Belluz, Alfonso Colombatti, and Paola Spessotto

PHASE IB RANDOMIZED, DOUBLE-BLINDED, PLACEBO-CONTROLLED, DOSE ESCALATION STUDY OF POLYPHENON E IN WOMEN WITH HORMONE RECEPTOR–NEGATIVE BREAST CANCER

DREGULATION OF XPC AND CYP A BY CYCLOSPORIN A: AN IMMUNOSUPPRESSION-INDEPENDENT MECHANISM OF SKIN CARCINOGENESIS
Weinong Han, Keyoumars Soltani, Mei Ming, and Yu-Ying He
ABOUT THE COVER

Gut-derived LPS promotes inflammatory hepatic injury and apoptosis by activating Toll-like receptor 4 (TLR4). Reduction of endotoxin using antibiotics regimen or TLR4 ablation in mice greatly attenuates hepatocyte apoptosis in a Con A–induced hepatitis model. Wild-type (wt) and TLR4 knockout (TLR4−/−) mice were injected with Con A intravenously and sacrificed 0, 6, and 20 hours thereafter. The micropictogram featured on the cover (magnification ×200) shows the apoptotic cells (brown) of liver sections from Con A-injected wt mice with antibiotic treatment using TUNEL assay in contrast to normal hepatocytes (green). Quantification of the apoptotic cells induced by Con A was significantly suppressed in both antibiotic-treated (P < 0.01) and TLR4−/− mice (P < 0.001; not shown) compared with control mice; this was further confirmed by the activation of caspase-3 and PARP demonstrating that the activation of TLR4-signaling pathway is important in Con A-induced hepatic injury in mice. See article by Lin et al. (beginning on page 1090) for more information.