**Perspectives**

1073 Genetic Driver Events in Premalignancy: LOH Validated for Marking the Risk of Oral Cancer  
Webster K. Cavenee
*Perspective on Zhang et al., p. 1081*

1075 Validation of LOH Profiles for Assessing Oral Cancer Risk  
Mark W. Lingen and Eva Szabo
*Perspective on Zhang et al., p. 1081*

1078 The Great Escape: Microbiotal LPS Takes a Toll on the Liver  
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**Research Articles**

1081 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. Gardiner, Huijun Jiang, Lang Wu, J. Jack Lee, and Miriam P. Rosin
*See Perspectives on p. 1073 and p. 1075*

1090 Gut-Derived Lipopolysaccharide 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
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1103 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

1115 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

1124 Failure Rates in the Hepatocellular Carcinoma Surveillance Process  
Amit 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

1131 An EMILIN1-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, Alfonco Colombatti, and Paola Spessotto

1144 Phase IB Randomized, Double-Blinded, Placebo-Controlled, Dose Escalation Study of Polyphenon E in Women with Hormone Receptor–Negative Breast Cancer  

1155 Deregulation of XPC and CypA 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.