Nrf2 Activation Protects against Solar-Simulated Ultraviolet Radiation in Mice and Humans
Elena V. Knatko, Sally H. Ibbotson, Ying Zhang, Maureen Higgins, Jed W. Fahey, Paul Talalay, Robert S. Dawe, James Ferguson, Jeffrey T.-J. Huang, Rosemary Clarke, Suqing Zheng, Akira Saito, Sukiriti Kalra, Andrea L. Benedict, Tadashi Honda, Charlotte M. Proby, and Albena T. Dinkova-Kostova

UCP2 Knockout Suppresses Mouse Skin Carcinogenesis
Wenjuan Li, Chunjing Zhang, Kasey Jackson, Xingui Shen, Rong Jin, Guohong Li, Christopher G. Kevil, Xin Gu, Runhua Shi, and Yunfeng Zhao

Ink4a/Arf-Dependent Loss of Parietal Cells Induced by Oxidative Stress Promotes CD44-Dependent Gastric Tumorigenesis
Ryo Seishima, Takeyuki Wada, Kenji Tsuchihashi, Shogo Okazaki, Momoko Yoshikawa, Hiroko Oshima, Masanobu Oshima, Toshiro Sato, Hirotoshi Hasegawa, Yuko Kitagawa, James R. Goldenring, Hideyuki Saya, and Osamu Nagano

Five-Year Cervical (Pre)Cancer Risk of Women Screened by HPV and Cytology Testing
Margot H. Uijterwaal, Nicole J. Polman, Folkert J. Van Kemenade, Sander Van Den Haselkamp, Birgit I. Witte, Dorien Rijkaart, Johannes Berkhoef, Peter J.F. Snoijers, and Chris J.M. Meijer

3,6-Dihydroxyflavone Suppresses Breast Carcinogenesis by Epigenetically Regulating miR-34a and miR-21
Xiaoli Peng, Hui Chang, Yeyun Gu, Junli Chen, Long Yi, Qi Xie, Jundong Zhu, Qianyong Zhang, and Mantian Mi

Effects of Metformin, Buforin, and Phenformin on the Post-Initiation Stage of Chemically Induced Mammary Carcinogenesis in the Rat
Zongtian Zhu, Weiqin Jiang, Matthew D. Thompson, Dimas Echeverria, John N. McGinley, and Henry J. Thompson

Anti-Müllerian Hormone Concentrations in Premenopausal Women and Breast Cancer Risk
Hazel B. Nichols, Donna D. Baird, Frank Z. Stanczyk, Anne Z. Steiner, Melissa A. Troester, Kristina W. Whitworth, and Dale P. Sandler

High-Density Lipoprotein-Cholesterol, Daily Estradiol and Progesterone, and Mammographic Density Phenotypes in Premenopausal Women
Vidar G. Hove, Hanne Prydenberg, Giske Ursin, Anita Iversen, Morten W. Fagerland, Peter T. Ellison, Erik A. Wist, Thor Egeland, Tom Wilsgaard, Anne McTiernan, Anne-Sofie Furborg, and Inger Thune

Dietary Patterns after Prostate Cancer Diagnosis in Relation to Disease-Specific and Total Mortality
Meng Yang, Stacey A. Kenfield, Erin I. Van Blarigan, Julie L. Baist, Howard D. Sesso, Jing Ma, Meir J. Stampfer, and Jorge E. Chavarro

Cucurbitacin B Alters the Expression of Tumor-Related Genes by Epigenetic Modifications in NSCLC and Inhibits NNK-Induced Lung Tumorigenesis
Samriddhi Shukla, Sajid Khan, Sudhir Kumar, Sonam Sinha, Mohd. Farhan, Himangsu K. Bora, Rakesh Maurya, and Syed Mushapara Meeran

Pilot Study on the Bioactivity of Vitamin D in the Skin after Oral Supplementation

LLPi: Liverpool Lung Project Risk Prediction Model for Lung Cancer Incidence
Michael W. Marcus, Ying Chen, Olaide Y. Raji, Stephen W. Duffy, and John K. Field
ABOUT THE COVER

Chronic inflammation induces histopathologic progression of the stomach epithelium leading to the development of metaplasia followed by gastric adenocarcinoma. Inflammation of the gastric epithelium, which produces high levels of reactive oxygen species (ROS), results in a gradual loss of parietal cells and their replacement with proliferative metaplastic cells, suggesting that the inflammation-associated ROS plays a role in the disruption of homeostasis of the gastric epithelium. However, the role of ROS and its downstream signaling in gastric carcinogenesis has remained unknown. The cover illustration depicts the phosphorylated (activated) form of p38MAPK (green) as well as parietal cells (H⁺,K⁺-ATPase; red) in normal stomach tissue exposed to the hydrogen peroxide in vitro (nuclei are counterstained in blue). As shown in the yellow signal (red and green overlay), the oxidative stress–dependent activation of p38MAPK is triggered selectively in parietal cells. For more information on the potential mechanisms underlying the oxidative stress–dependent parietal cells loss and consequent gastric carcinogenesis, see the article by Seishima et al. (beginning on page 492).