PERSPECTIVES

777 Navigating the Maize between Red Meat and Oncomirs
Patricia A. Thompson
See related article, p. 786

781 Selenium and Prostate Cancer Prevention: What Next—If Anything?
Merrill J. Christensen
See related article, p. 796

RESEARCH ARTICLES

786 Dietary Manipulation of Oncogenic MicroRNA Expression in Human Rectal Mucosa: A Randomized Trial
See related article, p. 777

786 Comparative Effects of Two Different Forms of Selenium on Oxidative Stress Biomarkers in Healthy Men: A Randomized Clinical Trial
See related article, p. 781

805 Inactivating Mutation in the Prostaglandin Transporter Gene, SLCO2A1, Associated with Familial Digital Clubbing, Colon Neoplasia, and NSAID Resistance
Kishore Guda, Stephen P. Fink, Ginger L. Milne, Neill Molyneaux, Lakshmeswari Ravi, Susan M. Lewis, Andrew J. Dannenberg, Courtney G. Montgomery, Shulin Zhang, Joseph Willis, Georgia L. Wiesner, and Sanford D. Markowitz

813 Rapid and Sustainable Detoxication of Airborne Pollutants by Broccoli Sprout Beverage: Results of a Randomized Clinical Trial in China

824 Inhibition of Endometrial Cancer by n-3 Polyunsaturated Fatty Acids in Preclinical Models

835 Myeloid Lineage–Specific Deletion of Antioxidant System Enhances Tumor Metastasis
Keiichiro Hiramoto, Hironori Sato, Takafumi Suzuki, Takashi Moriguchi, Jingbo Pi, Tooru Shimosegawa, and Masayuki Yamamoto

845 Loss of miR125a Expression in a Model of K-ras–Dependent Pulmonary Premalignancy

856 (+)-2-(1-Hydroxyl-4-Oxocyclohexyl) Ethyl Caffeate Suppresses Solar UV-Induced Skin Carcinogenesis by Targeting PI3K, ERK1/2, and p38
Do Young Lim, Mee-Hyun Lee, Seung Ho Shin, Hanyoung Chen, Joohyun Ryu, Lei Shan, Honglin Li, Ann M. Bode, Wei-Dong Zhang, and Zigang Dong

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Oxidative stress accelerates the pathogenesis of a number of chronic diseases, including cancer growth and its metastasis. Transcription factor Nrf2 (NF-E2–related factor-2) regulates the cellular defense system against oxidative stress. Systemic deletion of Nrf2 brings about an increased susceptibility to cancer metastasis, which is associated with aberrant ROS (reactive oxygen species) accumulation in myeloid-derived suppressor cells (MDSCs). However, it remains elusive whether cellular antioxidant defense system regulated by Nrf2 signaling in the myeloid lineage cells plays indispensable roles for metastatic cancer progression. In this study, myeloid lineage–specific Nrf2-deficient (N-MKO) mice are inoculated with mouse Lewis lung carcinoma cells and metastatic activity of the cells is examined. The cover micrograph depicts metastatic cancerous lesions in the lung tissue of tumor-bearing N-MKO mouse. Note that N-MKO mice develop multiple metastatic lung nodules, indicating that the Nrf2-deficiency in myeloid lineage cells accelerates cancer cell metastasis. See the article by Hiramoto and colleagues (beginning on page 835) for more information.