

COMMENTARY

- 171** Accelerating the Pace of Cancer Prevention-
Right Now



Graham A. Colditz and Karen M. Emmons

EDITORIALS

- 185** Immunomodulatory Effects of *Momordica charantia* Extract in the Prevention of Oral Cancer
Chinthalapally V. Rao

See related article, p. 191

- 187** The Conundrum of Omega-3 Fatty Acids in Cancer Prevention Studies: Which One? How Much? What Biomarkers?

Carol J. Fabian and Bruce F. Kimler

See related article, p. 203

RESEARCH ARTICLES

- 191** Bitter Melon Prevents the Development of 4-NQO-Induced Oral Squamous Cell Carcinoma in an Immunocompetent Mouse Model by Modulating Immune Signaling

Subhayan Sur, Robert Steele, Rajeev Aurora, Mark Varvares, Katherine E. Schwetye, and Ratna B. Ray

See related editorial, p. 185

- 203** A Randomized Multicenter Phase II Study of Docosahexaenoic Acid in Patients with a History of Breast Cancer, Premalignant Lesions, or Benign Breast Disease

Ayca Gucalp, Xi K. Zhou, Elise D. Cook, Judy E. Garber, Katherine D. Crew, Julie R. Nangia, Priya Bhardwaj, Dilip D. Giri, Olivier Elemento, Akanksha Verma, Hanhan Wang, J. Jack Lee, Lana A. Vornik, Carrie Mays, Diane Weber, Valerie Sepeda, Holly O'Kane, Margaret Krasne, Samantha Williams, Patrick G. Morris, Brandy M. Heckman-Stoddard, Barbara K. Dunn, Clifford A. Hudis, Powel H. Brown, and Andrew J. Dannenberg

See related editorial, p. 187

- 215** Pioglitazone Inhibits Periprostatic White Adipose Tissue Inflammation in Obese Mice
Miki Miyazawa, Kotha Subbaramaiah, Priya Bhardwaj, Xi Kathy Zhou, Hanhan Wang, Domenick J. Falcone, Dilip D. Giri, and Andrew J. Dannenberg

- 227** Adiposity, Inflammation, and Breast Cancer Pathogenesis in Asian Women

Neil M. Iyengar, I-Chun Chen, Xi K. Zhou, Dilip D. Giri, Domenick J. Falcone, Lisle A. Winston, Hanhan Wang, Samantha Williams, Yen-Shen Lu, Tsu-Hsin Hsueh, Ann-Lii Cheng, Clifford A. Hudis, Ching-Hung Lin, and Andrew J. Dannenberg

- 237** Genome-Wide Gene Expression Changes in the Normal-Appearing Airway during the Evolution of Smoking-Associated Lung Adenocarcinoma

Jacob Kantrowitz, Ansam Sinjab, Li Xu, Tina L. McDowell, Smruthy Sivakumar, Wenhua Lang, Sayuri Nunomura-Nakamura, Junya Fukuoka, Georges Nemer, Nadine Darwiche, Hassan Chami, Arafat Tfayli, Ignacio I. Wistuba, Paul Scheet, Junya Fujimoto, Avrum E. Spira, and Humam Kadara

CORRECTION

- 249** Correction: Whole-Genome Sequencing of Salivary Gland Adenoid Cystic Carcinoma

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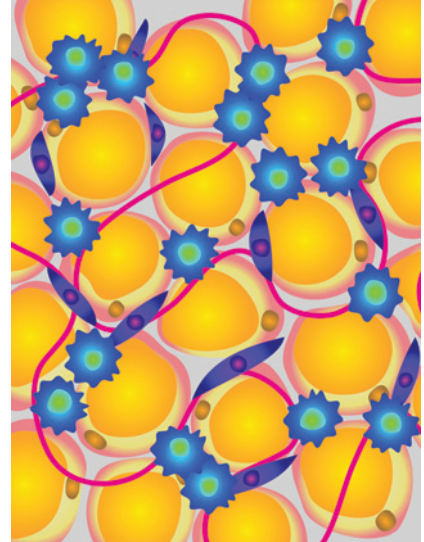
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Table of Contents

ABOUT THE COVER

Obesity-related inflammation is implicated in the development of breast cancer. Obesity-induced adipocyte hypertrophy, hypoxia and macrophage infiltration, and adipose stromal cell hyperplasia with increased local and systemic pro-inflammatory adipokines and cytokines fuel the process.

Preclinical and epidemiologic studies suggest that the inflammation resolving marine omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) reduce risk of breast cancer development. A key mechanism is thought to be replacement of arachidonic acid in cell membranes resulting in the formation of less inflammatory eicosanoids, as well as protectins and resolvins. In addition, EPA and DHA disrupt lipid rafts reducing activation of tyrosine kinase receptors such as EGFR. The dose of DHA or EPA combined or as single agents necessary for inflammation resolution or lipid raft disruption in humans is unclear; however, pre-clinical studies suggest that DHA may be superior to EPA for breast cancer prevention. Beginning on page 203, Gucalp and colleagues present results of a placebo-controlled trial in which 3 months of DHA at a dose of 2g/day did not impact expression of several inflammatory genes or prevalence of crown-like structures in benign breast tissue from overweight and obese high-risk women and breast cancer survivors. Potential explanations for these null results include the type and dose of fatty acid used, cohort heterogeneity, and the biomarkers selected, as discussed in an accompanying editorial by Fabian and Kimler on page 187.



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