OBITUARY

Peter Greenwald, Gary Stoner, Nancy Colburn, and Scott M. Lippman

PERSPECTIVE

Predicted for Greatness: 1994 Molecule of the Year—The DNA Repair Enzyme
Marianne Berwick
See article, p. 398

REVIEWS

Adherence to Endocrine Therapy in Breast Cancer Adjuvant and Prevention Settings
Rowan T. Chlebowski, Jisang Kim, and Reina Haque

Repurposing of Metformin and Aspirin by Targeting AMPK-mTOR and Inflammation for Pancreatic Cancer Prevention and Treatment
Wen Yue, Chung S. Yang, Robert S. DiPaola, and Xiang-Lin Tan

RESEARCH ARTICLES

Low Integrated DNA Repair Score and Lung Cancer Risk
Ziv Sevilya, Yael Leitner-Dagan, Mila Pinchev, Ran Kremer, Dalia Elinger, Hedy S. Rennert, Edna Schechterman, Laurence S. Freedman, Gad Rennert, Tamar Paz-Elizur, and Zvi Livneh
See commentary, p. 375

PI3K-AKT Signaling Is a Downstream Effector of Retinoid Prevention of Murine Basal Cell Carcinogenesis

Benign Breast and Gynecologic Conditions, Reproductive and Hormonal Factors, and Risk of Thyroid Cancer
Melissa Z. Braganza, Amy Berrington de González, Sara J. Schonfeld, Nicolas Wentzensen, Alina V. Brenner, and Carli M. Kitahara

LETTERS TO THE EDITOR

Cost-Effectiveness of a Genetic Test for Breast Cancer Risk—Letter
David G. Cox, Stéphane Ragusa, Lionel Pourtau, Lionel Perrier, and Suzette Delaloge

Cost-Effectiveness of a Genetic Test for Breast Cancer Risk—Response
Henry Folse, Richard Allman, and Tuan A. Dinh
ABOUT THE COVER

Perinatal exposure to bisphenol A (BPA) has been shown to cause aberrant mammary gland morphogenesis and mammary neoplastic transformation, though the underlying mechanism is poorly understood. Mammary glands of 21-day-old Balb/c mice were exposed to BPA by gavage (25 μg/kg/day) during puberty for 3 weeks to determine whether mammary stem cell (MaSC) function was altered leading to increased susceptibility to tumorigenesis through a stem cell-mediated mechanism. The cover art is a stylistic representation of the quantification of immunohistochemical staining patterns of FosB and c-Fos expression of regenerated ductal outgrowths in control or BPA-treated MaSCs. Pubertal exposure to the low-dose BPA increased lateral branches and hyperplasia in adult mammary glands and altered the function of MaSC from different age groups, causing early neoplastic lesions in their regenerated glands, suggesting that MaSCs are susceptible to BPA-induced transformation. The present study is the first to show that pubertal BPA exposure altered murine MaSC gene expression and function such that they induced early neoplastic transformation. See the article by Wang and colleagues (beginning on page 445) for more information.