2008

Prevention Journal Launched: *Cancer Prevention Research*

SCOTT M. LIPPMAN, MD, FOUNDING EDITOR-IN-CHIEF, 2008-PRESENT

As a renowned translational researcher, Dr. Lippman has been a principal investigator in numerous clinical trials for assessing cancer risk and for developing targeted drugs and personalized therapies. His research has focused on head, neck, and lung cancer, genetic drivers of cancer, predictive molecular signatures, and biomarkers for clinical response in solid tumors. He is currently co-investigator of an AACR Dream Team, funded by SU2C, involving molecular studies of lung cancer. In addition to maintaining an active clinical practice throughout his career, Dr. Lippman was chair of the Department of Thoracic/Head and Neck Medical Oncology at MD Anderson Cancer Center and is currently director of the Moores Cancer Center of the UC San Diego Health System.

Cancer Prevention Research

Contents

First Table of Contents

Inaugural Editorial

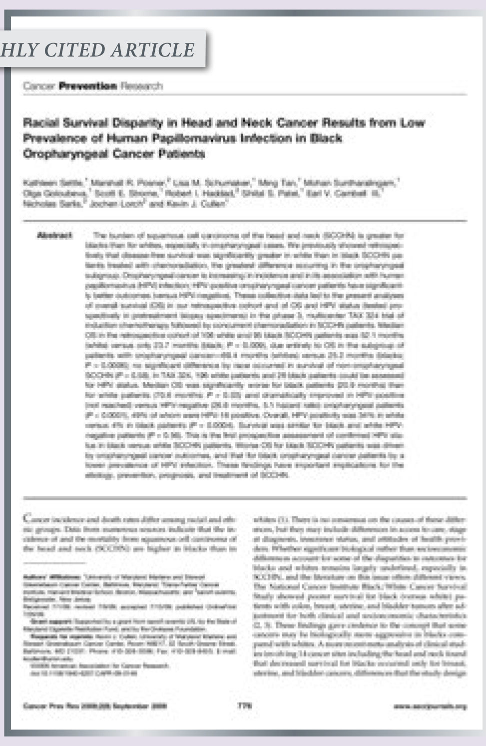
Letter from the Editor

2009

First Impact Factor: 6.000

Racial Differences in Survival of Head and Neck Squamous Cell Carcinoma Linked to HPV Positivity

HIGHLY CITED ARTICLE



Cancer Prevention Research

Racial Survival Disparity in Head and Neck Cancer Results from Low Prevalence of Human Papillomavirus Infection in Black Oropharyngeal Cancer Patients

Kathleen Settle, Marshall R. Posner, Lisa M. Schumaker, Ming Tan, Mohan Suntharalingam, Olga Goloubeva, Scott E. Strome, Robert I. Haddad, Shilal S. Patel, Earl V. Cambell III, Nicholas Sarlis, Jochen Lorch and Kevin J. Cullen

Cancer Prev Res 2009;2(9) September 2009 776 www.aacrjournals.org

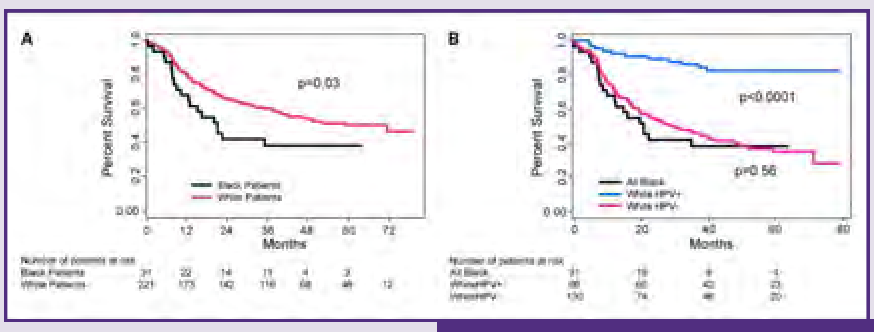


FIGURE 3. Overall survival by race and HPV-16 status in the TAX 324 trial. A, median OS by race in all patients: 70.6 mo [95% CI, 40.0-not reached (NR)] for white patients versus 20.9 mo (95% CI, 12.4-NR) for black patients (log-rank test $P = 0.03$). B, median OS by race and HPV status (log-rank test $P < 0.0001$): NR for white HPV-positive (HPV+) patients; 30.1 mo (95% CI, 19.7-42.0) for white HPV-negative (HPV−) patients; and 20.9 mo (95% CI, 12.4-NR) for black patients. The difference in survival between all black patients and HPV-negative white patients was not significant ($P = 0.78$). Of 32 black patients with an available biopsy, only one was HPV positive. The difference in survival between HPV-positive white patients and all other patients was highly significant ($P < 0.0001$).

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2010

Raloxifene and Tamoxifen Are Good Choices for Postmenopausal Women with Elevated Breast Cancer Risk

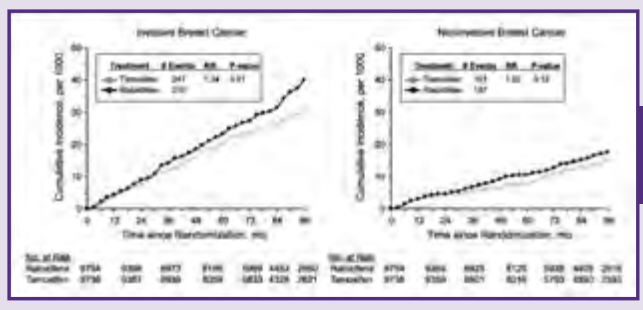

FIGURE 1. Cumulative incidences of invasive and noninvasive breast cancer.

2011

Oral Prostacyclin Analogue Efficacious Chemoprevention for Former Smokers but Not for Active Smokers

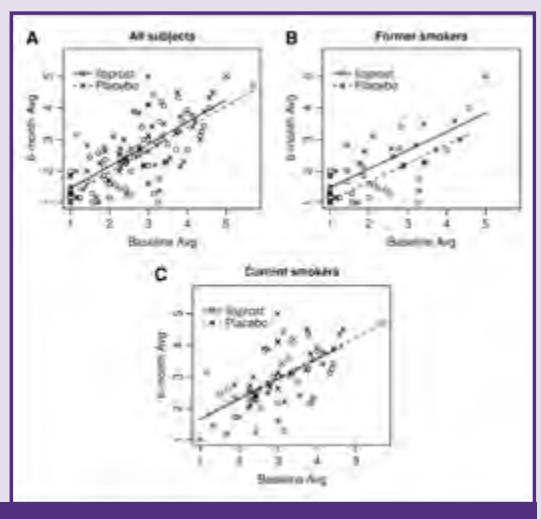

FIGURE 2. Six-month histology as a function of baseline average histology in all subjects (A), former smokers (B), and current smokers (C) comparison of average histology measures on initial and follow-up bronchoscopy of subjects completing the trial (60 iloprost subjects and 65 placebo subjects). Illustrates consistent improvement across the entire histologic spectrum in former smokers receiving iloprost ($P = 0.010$) and a lack of effect in all subjects ($P = 0.210$) and current smokers ($P = 0.743$).

2016

Precision Medicine and Immuno-oncology Transform Cancer Prevention

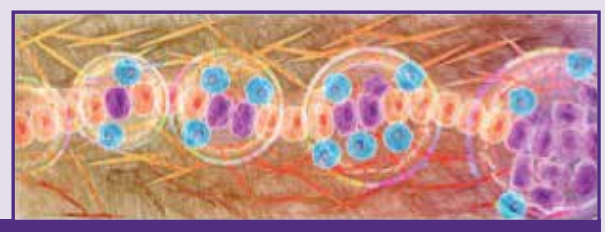

FIGURE 1. The molecular alterations associated with early pathological steps preceding the development of invasive carcinoma have not been well characterized. A Pre-Cancer Genome Atlas (PCGA) is needed both to support the collection and molecular profiling (circus plot) of premalignant lesions (purple cells) to identify the sequence of initial driver events that cause normal cells (orange cells) to acquire cancer hallmarks that enable lesions (purple cells) to progress to fully invasive carcinoma, including the critical "additional genomic events" (e.g., checkpoint/tumor suppressor loss or other co-activating event) that transform premalignancy (purple cells in the fourth circle to the right) to cancer (far right). In addition to defining the sequence of genomic driving events in specific neoplastic sites, characterizing the premalignant inflammatory microenvironment, including the contribution of the stroma and immune cell (blue) regulation, will provide a better understanding of the selective forces that determine which molecular drivers give an evolutionary advantage to drive premalignant lesions to become invasive cancer. This figure is modified in part from Campbell et al. (21).

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