IN THIS ISSUE
Highlighted research articles 377

NEWS IN BRIEF
Important news stories affecting the community 380

NEWS IN DEPTH
Q&A: Christopher Wild on Global Tobacco Use 383
J&J Partners with Yale to Share Trial Data 384

RESEARCH BRIEFS
Selected highlights of recent articles of exceptional significance from the cancer literature 385

ONLINE
For more News and Research Watch, visit Cancer Discovery online at http://CDnews.aacrjournals.org.

REVIEW
Contribution of p53 to Metastasis 405
E. Powell, D. Piwnica-Worms, and H. Piwnica-Worms

RESEARCH BRIEFS
CD74–NRG1 Fusions in Lung Adenocarcinoma 415

Pre cis: CD74-NRG1 fusions are observed in invasive mucinous lung adenocarcinomas and drive cell transformation via activation of ERBB3–PI3K–AKT signaling.

Response of BRAF-Mutant Melanoma to BRAF Inhibition Is Mediated by a Network of Transcriptional Regulators of Glycolysis 423

Pre cis: MYC, HIF1α, and MONDOA act downstream of BRAF to regulate glycolysis and mediate melanoma cell sensitivity to BRAF inhibition by vemurafenib.

In The Spotlight
Metabolic Dysregulation in Melanoma: Cause or Consequence? 390
R. Haq
See article, p. 423

Unlikely Suspects Identified in Neuroblastoma Conspiracy 392
R. Bernards
See article, p. 434

Germline Polymorphisms in RNF31 Regulate Linear Ubiquitination and Oncogenic Signaling 394
P. Grumati and I. Dikic
See article, p. 480

Prospective
Oncology Drug Discovery: Planning a Turnaround 397
C. Toniatti, P. Jones, H. Graham, B. Pagliara, and G. Draetta

Q&A: Christopher Wild on Global Tobacco Use

J&J Partners with Yale to Share Trial Data

Pre cis: CD74-NRG1 fusions are observed in invasive mucinous lung adenocarcinomas and drive cell transformation via activation of ERBB3–PI3K–AKT signaling.

Response of BRAF-Mutant Melanoma to BRAF Inhibition Is Mediated by a Network of Transcriptional Regulators of Glycolysis

Pre cis: MYC, HIF1α, and MONDOA act downstream of BRAF to regulate glycolysis and mediate melanoma cell sensitivity to BRAF inhibition by vemurafenib.
Fernandez-Cuesta and colleagues identified recurrent fusions between CD74 and the exons encoding the EGF-like domain of the neuron-specific neuregulin 1 (NRG1) III-β3 isoform in invasive mucinous lung adenocarcinomas that lack common kinase driver mutations. The CD74–NRG1 fusion generates a membrane-bound protein that exposes the EGF-like domain of NRG1 on the extracellular surface, which creates a ligand for ERBB2–ERBB3 heterodimers and promotes oncogenic transformation by activating the PI3K–AKT pathway downstream of ERBB3. These findings implicate CD74–NRG1 as an oncogenic driver in lung adenocarcinomas and suggest that the ERBB3–PI3K–AKT pathway may be a therapeutic target in the invasive mucinous subtype, which currently lacks effective treatments. For details, please see the article by Fernandez-Cuesta and colleagues on page 415.